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THE DUTIES AND RESPONSIBILITIES OF THE CIVIL SURGEON WHEN CALLED TO ACTIVE MILITARY SERVICE *

BY GEORGE EMERSON BREWER, M.D.
OF NEW YORK, N. Y.

IT has always been the custom of the American Surgical Association to allow the president a large measure of latitude in selecting the subject of his inaugural address; and while in the majority of instances the subjects presented have been strictly professional, in that they have dealt with some live surgical problem, on not a few occasions the topics chosen have been of historic or biographic interest, or have presented thoughts on medical education, professional ethics, public health, or military preparedness.

That the interest of the surgical profession, here and elsewhere, has been acutely centred, during the past few years, upon the various military phases of our art, is evidenced by the large number of communications presented at our last meeting which dealt with the treatment of battle casualties, and with their associated infections; or the new and improved methods employed in the care of those who had been injured by the novel and cruelly destructive agencies of modern warfare, which by their diabolical ingenuity, have far outclassed in their mutilating effects anything which the world has ever known; and if it were not for the fact that in our present session a part of our program is made up of papers dealing with these same topics, I feel that I might have yielded to the strong impulse to present to you on this occasion some personal observations and experiences in front area work.

I have, however, elected to speak this morning upon two matters not strictly germane to either the scientific or practical aspects of our professional work, but which I venture to hope may be of interest. The first is a report on the activities of the Fellows of this Association in war work, and the other the duties and responsibilities of the civil practitioner in time of war, or perhaps, to state it more definitely, the relationship between the Army Medical Corps and the trained civil surgeon when called to active military service.

When we consider that our Association embraces in its membership the great majority of the recognized leaders of surgery in America, that owing to the conditions of fellowship over 90 per cent. of its active members are well above the military age, and were under no moral obligation

* President's Address before the American Surgical Association, May 3, 1920.

to enter the Government service, it is a matter of genuine pride and satisfaction to know that over 85 per cent. of our Fellows promptly volunteered and gave generously of their time and effort to the cause. I know of no other body of men of equal standing in the community who gave so much and at a greater sacrifice of personal interests.

It is fitting, therefore, that some record of their activities be preserved and made a part of our Association Archives, not only to record the historic facts, but to furnish an example to those who may follow, in the event of another and similar national emergency.

I have, therefore, attempted to collect all available data as to the service rendered by members of this Association, and will give you a brief summary of the result.

The total number of Fellows, active and senior, who offered their services to the Government or engaged in actual war work was 154, the average age of these was fifty-six and one-half years. It is worthy of note that of the 33 senior Fellows, 25 volunteered or engaged in active service, the average age of this group being seventy.

Number holding commissions in the U. S. Army, including two contract surgeons	96
Number holding commissions in the British Army	4
Number holding commissions in the Canadian Army	6
Number holding commissions in the French Army	1
Members of the Reserve Corps, who volunteered but were not called to active duty or were rejected on account of age or physical disability	6
Volunteer surgeons serving with the British Army previous to 1917	5
Volunteer surgeons serving with the French Army previous to 1917	13
Commissioned officers in the U. S. Navy	9
Fellows serving in the Red Cross Society	13
Fellows serving in the Medical Advisory Committee of the Council of National Defense	9
Enrolled in the Volunteer Medical Service Corps	11
Serving in local military or examining boards, State organizations or detailed to give instruction in military surgery	36

Of the 107 who held commissions in our own or one of the Allied armies, there were:

Brigadier-generals	1
Colonels	25
Lieutenant-colonels	37
Majors	31
Captains	7
First lieutenants	4

In two instances the rank was not mentioned.

Of these, 57 served in France with the A. E. F., 48 in the United States, 11 in the French or British armies.

As a number of officers served at different times in two or more of the Allied armies, and in different organizations in our own army, the totals in this and the following groups obviously will not correspond to the exact number of commissioned officers.

Number serving in base or general hospitals	55
Number serving in evacuation hospitals or casualty clearing stations	12
Number serving in mobile hospitals	3
Number serving in camp hospitals	6
Number serving as heads of surgical teams	19
Number serving as consultants	26
Number serving at Headquarters at Washington	8
Number serving at Headquarters, A. E. F.	2
American delegates to Interallied Surgical Conferences in Paris	6
Fellows receiving one or more promotions	77
Fellows receiving decorations	22
Fellows receiving citations	10
Fellows mentioned in dispatches	4
Fellows serving in front area during one or more of the great battles	47

This is but a brief and incomplete statement of the actual service rendered by our members, and is introduced here, merely to give a general idea of the important and responsible positions they held during the World War. I have prepared a fuller record of the services rendered by each of the Fellows, which will be appended to this address and published in the transactions.

Let us now consider my second topic, *The duties and responsibilities of the trained civil surgeon when called to active military service.*

That he has a definite responsibility can not be questioned when we consider that in time of war or active mobilization, the Medical Corps of the Army is of necessity greatly augmented by the enlistment of men from the reserve corps and from civil practice. To illustrate the extent of this augmentation, allow me to call attention to the fact that at the time of our declaration of war, the Medical Corps of our Army consisted of less than 500 medical officers. At the time of the armistice the number of commissioned medical officers was considerably over 30,000, which indicates that at that time more than 59/60, or 98.3 per cent., of the medical service of the Army was rendered by civilian practitioners, including surgeons, internists, sanitary experts, laboratory workers, and other specialists.

The chief function of the Medical Corps of an army is to render the best possible sanitary service to the troops, to keep them in the best physical condition, and to provide individual care and skilled professional attention for the sick and injured.

During peace this is not difficult, the number of sick and injured is small, the skill and experience of the officers are well known, and they easily can be assigned to duties which they are qualified to assume. In time of war or active mobilization, however, the problem is far more difficult, for it necessitates a complete reorganization of the corps, the assimilation of thousands of new men who are ignorant of army routine, and whose professional qualifications are to a large extent unknown.

When we consider the magnitude of the problem and the difficulties under which the Surgeon General labored during our recent mobilization, the marvel is that so much in the way of efficient organization was accomplished.

Laying aside for the moment the activities of the medical, sanitary, laboratory, and special departments, what was the chief surgical problem to be solved? I take it you will all agree with me that it was to render prompt and skilled surgical care to the man wounded in battle. The man who has the courage, patriotism, and determination to go into battle and give every ounce of energy and strength which he possesses to defeat the enemy, who cheerfully faces death and the chance of mutilating and disabling injury, is certainly entitled, when wounded, to the best surgical skill which his Government is able to provide. If he receives anything less than this, he is not being treated fairly, or, to use a commonplace expression, he is not getting a square deal.

How best can this be accomplished? To what extent was this accomplished in our own army during its participation in the great war? In answering the first question, I believe that one of the most important factors is to avoid misfits. By that I mean men who are assigned to duties they are not qualified to fulfill, or retained in such positions after their unfitness has been demonstrated. To obtain the best results only men of adequate surgical training and of large experience should be selected as operating surgeons in advanced hospitals where the wounded receive their first surgical treatment; and the work of these men should under no circumstances be hampered or interfered with by men of higher rank, but without skill, training, or experience in modern surgical procedures. Likewise in the base hospitals to which the wounded are quickly transferred from the front area, there should be a sufficient number of trained surgeons to oversee and direct the work of a larger number of junior officers, younger men, who have had at least some preliminary training in modern surgical technic.

That this ideal arrangement has not generally been carried out in the past will be evident to any one who will take pains to read the medical and surgical histories of any of the great wars of modern times. In the majority of instances these failures have been due, not to indifference on the part of a Government to the fate of its wounded soldiers, but to misfits in the professional personnel; expert surgeons who are assigned to purely executive duties, medical men who are assigned to responsible surgical posts, oculists, aurists, dermatologists, and X-ray operators who are obliged to work in medical or surgical wards, or in some specialty not their own, when their skill is urgently needed elsewhere; men well trained in laboratory methods but without experience in clinical work, obliged to give their entire attention to clinical problems.

All of these misfits I have personally observed in innumerable instances, have watched their bungling unproductive work, and have listened to a recital of their many efforts made through various channels to be given work which they felt themselves competent to carry out. I am not now speaking alone of our army, but of experience gathered while serving with the French or British forces; and I think it only fair to state

that, while numerous examples could be pointed out of such misfits in our own organization, my observations lead me to believe that there were fewer such instances in the A. E. F. than in the other Allied forces, and that it was far easier with us to effect satisfactory transfers, largely through the cordial relations which existed between our Chief Surgeon and the commanding officers of our sanitary groups.

One of the causes of this difficulty in the past has been due to the fact that ranking officials in the administrative bureaus, while perhaps possessing expert ability in executive matters, fail to recognize how highly specialized medicine has become during the past half century, to realize that the battle casualties of modern warfare present, in perhaps the majority of instances, the gravest of surgical problems, or to appreciate how utterly futile it is to expect these problems to be successfully met by men without or with but limited training or experience. As one British officer stated, "The men at Headquarters feel that every man possessing a medical diploma is capable of any and all kinds of professional work."

Another and perhaps the chief cause of misfits is the appointment of men to positions of grave professional responsibility on account of rank or previous service rather than professional qualifications. But the question is asked, How could this be otherwise without demoralizing the morale of the corps? The Army is an organization into which men enlist for life. They begin at the bottom and gradually work their way to the higher ranks by years of painstaking conscientious work, and when a man of mature age, after fifteen or twenty years of faithful service, reaches the rank of major, lieutenant-colonel, or colonel, is he not qualified to accept grave responsibility, and in the event of war, is it fair that he should be cast aside and replaced by a civilian who has never served in the Regular Army, and knows nothing of military routine?

Let us meet the issue squarely, and consider it from every angle, bearing in mind the paramount duty of the Government, which is to render to the wounded soldier the best possible surgical skill.

In the Regular Army the medical officer at the time of his enlistment is a highly qualified man, but with limited experience. During the first eight or ten years of his service, he is assigned to one or several military posts, where he has the care of a limited number of physically fit men, and the families of the officers. Between these assignments he may be stationed at a military hospital where he may have purely administrative duties, or may serve in medical or surgical wards. At other times he may have bureau work at Washington or at some divisional headquarters. As he advances in rank, he is given more responsible duties of an administrative character with a progressive diminution in actual professional work. As one major expressed it to the writer, "I have been seventeen years in the service. During my first six years my work was largely professional, during the next four or five years it was about equally

divided between professional and administrative duties; and for the past seven years I have had practically no professional responsibility."

Certainly the army in peace time is not an ideal school for the training of surgeons, and while there are, of course, notable exceptions, of men who have had long periods of service in hospitals on account of special aptitude for medical, surgical, or special work, the opportunities for intensive surgical training are few, and the majority of army surgeons who have served perhaps fifteen or more years, and have reached the rank of major or lieutenant-colonel, while they may be expert administrative officials, can not be regarded as highly qualified modern surgeons. Such an experience can not qualify even the most gifted man to meet the emergencies or assume the grave responsibilities of treating battle casualties.

With the recent civilian graduate who has chosen surgery as his special life work, it is entirely different. From the time he leaves the medical school his energy is directed in a single channel. He passes through the positions of surgical interne, house surgeon, or resident surgeon, outpatient surgeon, assistant surgeon to the wards, associate or junior surgeon; and at the age when our military surgeon reaches the rank of major or colonel, the civilian practitioner if capable and industrious has reached the goal of his ambition, is an attending surgeon to some hospital, a position gained by fifteen or twenty years of continuous intensive surgical training.

If in the necessary reorganization of the army medical corps in time of war, the general policy were followed of selecting highly qualified civilian surgeons, but without army experience, to positions of purely professional responsibility, where a knowledge of army administrative methods is not essential; and of the highly qualified members of the regular corps to positions of high administrative command, where their knowledge and experience are most needed, it would provide promotion and dignified positions for all the capable ranking men of the service, and would in no way tend to demoralize the morale of the department. It would also prevent in a large measure the misfits to which I have alluded, and would be the greatest factor in providing for the wounded soldier the highest type of surgical skill. This was the general policy in our army during the recent war; but with a less enlightened and broad-minded Surgeon General it might not be the policy in a future war. Moreover, the line was never definitely drawn between administrative and professional control, and most of us who served in France saw examples of men of high rank holding executive positions, issuing orders which if carried out to the letter would have sadly interfered with the orderly carrying on of modern surgical procedures.

This brings me to the second factor in accomplishing the highest degree of professional service to the wounded man; and that is the plan of dual control in all hospitals and all organizations in which the medical department has important activities. I realize that the term dual control

of any military formation will be said to be a blow at the very foundation of military discipline. Yet I venture to approve the plan for the reason that I believe it to be fundamentally sound, that it was first suggested and put into operation by our own Surgeon General, and also for the reason that I think it can be shown that by a reasonable interpretation it will not affect or interfere with military discipline in the slightest degree.

It will be recalled that long before we entered the war the Surgeon General authorized the organization of fifty Red Cross Base Hospitals, with the understanding that in case of war they would be taken over by the War Department and made an integral part of the army. The plan of organization was to supply for each a commanding officer appointed from the Medical Corps of the Regular Army, who would have complete administrative and disciplinary control of the unit; and a director who would be responsible for the actual care of the patients. By this plan these hospitals were placed on the same basis as our own best civil hospitals in which the Board of Trustees or administrative department is entirely separate from the professional, and in no way interferes with or attempts to dictate the scientific activities of the professional staff; but at all times is in absolute control, as they have the power of appointment and removal.

The success of the plan was, I think, generally admitted. In the unit to which I was attached and in a number of others in which there was a reasonable coöperation between the commanding officer and director, there was not the slightest friction, and no question of authority was ever raised; the director recognized that the commanding officer was his superior officer, and the commanding officer recognized the professional responsibilities of the director and never interfered with the clinical work of the unit.

In the late autumn of 1917 orders were sent from Washington to the A. E. F. to organize a group of professional consultants, to take over the responsibility of the care of patients in the various divisions as they became ready for active duty. Without going into detail in regard to the organization of this group, with which you doubtless are all familiar, I may briefly state that there was a chief consultant in surgery, a chief consultant in medicine, and a chief consultant in the laboratory specialties. Under each of these departments there were a number of subdivisions, those in surgery being: General surgery, orthopedics, urology, otology and laryngology, ophthalmology, facio-maxillary surgery, neurological surgery, and experimental surgery.

Special divisional consultants were first appointed in general surgery, orthopedic surgery, and urology. Later consultants in medicine, neurology, and some of the other specialties were appointed to divisional, corps, and army headquarters. At a still later period, surgical consultants were sent to a number of the large base hospital centres, where they would direct and supervise the professional work in the various hospital units.

Shortly after the creation of the Consulting Board, the Chief Surgeon of the A. E. F. authorized the chief consultant in surgery to organize surgical teams for active service at the front, relying upon his judgment in the selection of the officers to head each team. More than one hundred such teams, representing the best surgical talent in the overseas army, were organized and sent to the evacuation and advanced hospitals in the three or four great battles in the summer and autumn of 1918. During this active period the chief consultant arranged frequent conferences at which a number of the front area divisional or corps consultants took part, and at these meetings general rules regarding the surgical care and operative treatment of the various types of battle casualties were freely discussed and adopted, and instructions issued to all consultants to be transmitted to the heads of the surgical teams. These instructions in general conformed with the suggestions issued by the Inter-Allied Surgical Conference, modified to some extent by the experience of our own men. While few of our operating surgeons heading surgical teams had had any experience in the treatment of battle casualties, they were nearly all men of experience, with adequate surgical training, and in not a few instances had had opportunities to observe the best type of military surgical procedure in some of the best French, English, and Belgian hospitals, as well as in our own Evacuation Hospitals Nos. 1 and 2, which were organized early, in quiet sectors, and in which some of our most experienced men were operating and giving instruction in the technic of modern military surgery. This and the fact that nearly all of our consultants in the front area had had previous experience in the British, French, or Belgian armies, made it possible for our advanced hospitals to render such excellent service during the periods of great activity.

While I would not have you believe that this advanced service in any way approached perfection, I think I can truthfully say that, taken as a whole, it was better than I had previously observed in any sector of the same size during a period of active military operations. When failure or disaster occurred, it was not the result of lack of skillful operative measures, but was rather due to overcrowding, delayed transportation and absence of forethought in providing adequate hospital accommodations, teams, nurses, and supplies. In other words, it was due to administrative rather than professional errors.

From this brief statement regarding the general plan of dual control, I feel that you will all agree that it represented a wise and honest attempt on the part of our Surgeon General to improve the quality of the service rendered to our wounded men. That it was not more satisfactory in its operation was due to a number of circumstances.

In the first place the plan should have been carefully considered and its organization thoroughly effected before we entered the war.

Specific regulations should have been adopted defining the duties of

the administrative and professional chiefs, so that there should be no conflict of authority.

Copies of these regulations should have been sent to all commanding officers and to chief divisional, corps, and army surgeons, well in advance of assigning consultants to duty.

The official orders to consultants should have been uniform, explicit, and delivered at the time of appointment.

Had this been done, the status of the consultant would have been established. As it was, the arrival of the consultant at divisional headquarters was often the first intimation the chief divisional surgeon had that such a position had been created; and if, as frequently happened, the consultant's orders were not explicit, his presence was resented and looked upon as an attempt to destroy the prestige and undermine the authority of the divisional chief. In a few instances this resulted in open hostility and complete lack of coöperation, rendering the consultant's position extremely trying, and greatly interfering with his usefulness.

Lack of uniformity or great delay in issuing orders was a frequent source of misunderstanding. On more than one occasion I was sent to various parts of the line without any written orders. At other times my orders would read, "Will proceed to this or that headquarters and report to the divisional or corps surgeon." On other occasions my orders would be explicit and state, "Will proceed to Division ——, will supervise and direct the surgical work in all divisional hospitals, and all evacuation hospitals assigned to or situated in that sector; operate himself when deemed advisable; and in general carry out the orders of the Chief Surgeon, A. E. F., and chief consultant in surgery," thus clearly indicating that in professional authority he was responsible only to the chief consultant or the chief surgeon of the expeditionary force.

When we consider that the plan was an entirely new one, was not mentioned in the manual, was hastily considered in Washington, and transmitted to the A. E. F. without definite instructions, that no definite and uniform rules were established for its operation, that orders were not uniform, were frequently vague, and often greatly delayed, and that the line officers were generally left in complete ignorance of the plan and the status and authority of the consultants; it is a marvel that it succeeded as well as it did. In my opinion, its limited success was due to the vision and broad-minded attitude of General Ireland and his able assistants at the Chaumont Headquarters, to the honest efforts of the consultants themselves, and the hearty and intelligent coöperation of the majority of the regular officers.

I am thoroughly convinced that had the war lasted another six months, and had General Ireland's wise policy been continued in the A. E. F., after his promotion to the position of surgeon general, all obstacles would have been overcome, and the American system of professional control would have been declared an unqualified success.

In these few remarks I have attempted to answer briefly the two questions propounded in the opening paragraphs of this part of my address, but there is now another and more important question to be considered, and that is, what in the light of our past experience can be done now to insure better treatment for our wounded men in the event of another war, or to provide better care of our mobilized men if laws should be enacted authorizing universal military training.

In answer to this question permit me to say that, in my opinion, we can not do better than to adopt in principle the plan of dual control proposed by our surgeon general.

I sincerely believe, as stated above, that it is fundamentally sound, and the only plan that will insure to the wounded the highest degree of professional service.

That it was far from perfect in its operation during the late war, we will admit; but its imperfections and disadvantages were trivial in comparison to the advantages it presented, and easily could be remedied by more perfect organization. While doubtless it would be desirable to modify to some extent the regulations in force during the war, the plan should be essentially the same, and should embody the appointment of a chief consulting surgeon, a man of the broadest experience chosen from the civilian profession, possessing the highest qualities of surgical judgment and technical skill, who has had previous military experience, and who is also possessed of organizing ability. His headquarters should be in the office of the surgeon general, and to him all questions dealing with the actual surgical care of patients should be referred for his expert advice. That this chief consultant should have a number of deputies or assistant chief consultants, also men of conspicuous surgical ability and large experience. One of these to be assigned to headquarters of each army to coöperate with the administrative chief of the medical service. Under this deputy chief consultant there should be an adequate number of active consultants who could be assigned to corps or divisional areas in charge of the surgical work of the various hospital units, and who would be responsible through the army and chief consultant to the surgeon general for the carrying out of the most approved modern surgical methods in the treatment of the wounded men. This plan would obviously include a similar organization of the departments of internal medicine and sanitary service, with as many subdivisions of each as would be found to be necessary.

This, I beg you to bear in mind, is but the expression of my own personal view. I do not suggest it as a plan to be blindly followed or adopted without the fullest and freest discussion, in a conference composed of regular army officials and civilian practitioners who have had actual military experience; but I think that the time is now ripe for such a conference and interchange of views, and that now in time of peace some plan should be worked out to give to the American Army the best

professional service which it is possible to organize, which will insure to the sick and injured soldier the same degree of professional skill that he could receive in the best organized and equipped civil hospital in the country.

While such a plan would be an emergency measure, and while the consulting staffs would be members of the reserve corps, and not on duty in peace time, it should be organized now, down to the last detail, so that in the event of our country facing another military crisis, it would not be necessary to devise ways and means to meet the emergency in haste, during a period of stress, excitement, anxiety, and feverish activity.

I do not feel that we as an association of civilian surgeons should apologize for considering this problem, or should hesitate to offer to the military authorities our suggestions on a subject which so deeply concerns us. If in time of war we are to bear such a large proportion of the burden of responsibility, we are certainly entitled to a voice in the making of plans and regulations under which we are to assume it. It is for this reason that I have brought this matter to your attention. I have been so overwhelmingly impressed with the importance of this subject, that it seems to me it would be a gross neglect of duty if I were to have chosen for this address another and perhaps more conventional topic.

My message comes from the heart of one who has been an eye witness to the monstrous and cruel toll in mortality and wrecked lives which war inflicts upon the flower of the youth and promising manhood of a nation; and I urge upon you, as members of the most distinguished group of surgeons in America, to be prepared, if the opportunity is offered, to cooperate with our Government officials in proposing some enlightened plan which will raise the standard of our military medical service to a plane never before reached in the world's history.

OBSERVATIONS ON EMPYEMA*

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surgeon to the episcopal hospital, associate in surgery in the university of pennsylvania

At the meeting of the American Surgical Association, twenty-six years ago, my father opened the discussion on empyema. He laid down the following six propositions for discussion:

1. No operation is justifiable
   (a) Unless the presence of pus is certain.
   (b) Unless thorough treatment by medicinal agents, blisters, etc., has failed.
   (c) Or unless the symptoms, dyspnœa, etc., are so urgent as to demand immediate relief.
2. The first operation should consist of simple aspiration with antiseptic precautions.
3. That when the fluid has partially reaccumulated, as it almost certainly will do if purulent, incision and drainage should be practiced
4. That drainage is best effected by making two openings, one at the lowest available point, and carrying a large drainage tube through the cavity from one opening to the other.
5. That drainage should be supplemented by washing out the cavity with mild antiseptic fluids. When the lung has expanded and discharge has nearly ceased, the tube should be shortened, the upper opening being allowed to heal and the tube being then gradually withdrawn through the lower opening.
6. That when the lung is so bound down by adhesions that it cannot expand, resection of one or more ribs should be practiced (Estlander's operation, so called), in order to allow collapse of the chest wall and to promote healing by bringing the costal and visceral layers of the pleura into contact. The more extensive operations of Schede and Tillmans, while probably justifiable in exceptional cases, are not to be recommended for general employment.

The mortality following operation for empyema at that time averaged from 20 to 30 per cent., and such it still remained in the hands of the average surgeon during the generation which has elapsed since 1894. It is true that in most of the fatal cases the operation is not to blame for the fatality, but merely fails to save life; and that death is due to the underlying disease—empyema.

It is my object in this discussion to see, if possible, in what ways we have made an advance over the teachings and practices of a generation ago.

In the first place, I call attention to the *first proposition*, "That no operation is justifiable except under one of these conditions," *viz.*, (*a*) that the presence of pus is certain, (*b*) that medical treatment has failed, (*c*) or that the symptoms are very urgent. Is any one at present ready to go beyond this, and adopt operation under any other circumstances? Do we not always receive our patients from the physician, whose treat-

* Read before the American Surgical Association, May 3, 1920.

ment has failed? True it is that the physician almost invariably has made one or more punctures of the chest, with or without success in locating pus, before he calls the surgeon into consultation; and that similarly even when pus has been found he is often loath to turn the patient over to the surgeon until he finds that the pus does reaccumulate; and even under the latter circumstances he may persist in the attempt to cure the patient without surgical assistance, by means of antiseptic injections into the pleural cavity. So eminent and able a surgeon as John B. Murphy, it will not be forgotten, advocated and practised injections of a 2 per cent. solution of formalin in glycerine for the cure of empyema; but no statistics of this treatment, showing its results, have ever been

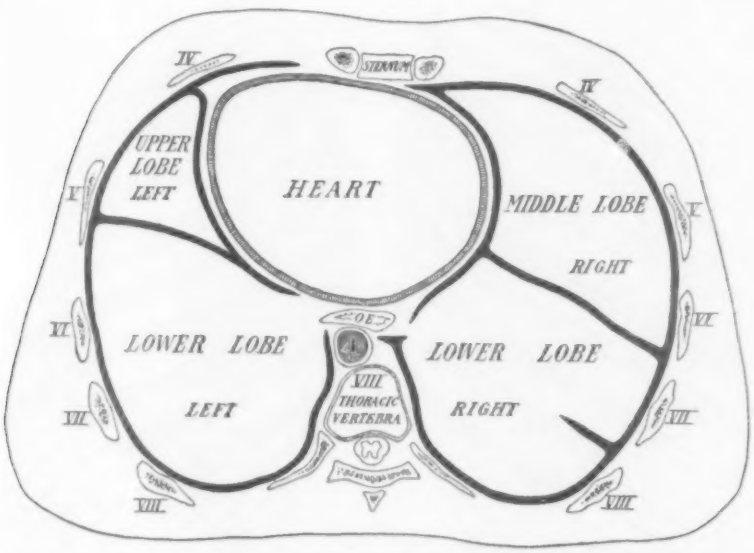


FIG. 1.—Cross-section of thorax at level of eighth thoracic vertebra. Pleural cavities outlined in black.

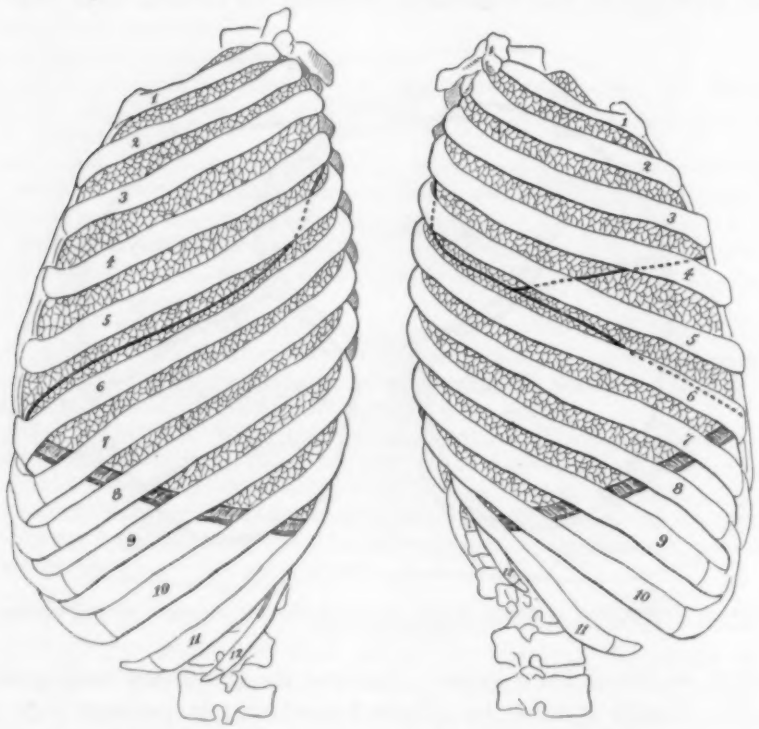
published, so far as I am aware. Nor has the treatment been generally accepted; though it must be acknowledged that in perhaps 2 or 3 per cent. of cases in which the first aspiration shows only sero-pus, not frank pus, it is possible that a cure may be secured either by repeated aspiration alone, or by aspiration followed by injection of formalin-glycerine solution, flavine, etc. So much, therefore, for the contingency which renders operation proper when medical treatment has failed.

Now if the symptoms are very urgent, that is, if there is extreme dyspnœa, cyanosis, bulging interspaces, etc., every one admits the propriety of aspirating the chest whether or not it is certain beforehand that pus, not serum, is the cause of the symptoms. Finally, if the presence of pus is certain, comparatively few surgeons deny the propriety of attempting to evacuate it.

But one may rejoin, how is it possible to be certain that pus is

present except by aspiration? Are the clinical history, the physical signs, the X-ray findings, and the symptoms always sufficient to render a diagnosis certain? To this I think we must reply in the negative; but I submit we must at the same time recognize that there are cases where the presence of pus is so nearly certain, even when it cannot be located by puncture, that it is not only justifiable but imperative for the surgeon to open the thorax, search for the encapsulated empyema, and effect its evacuation and drainage.

Some years ago, before the Section on General Medicine of the College of Physicians of Philadelphia, I presented[1] a plea for exploratory



FIGS. 2 AND 3.—Interlobar fissures and their relation to the ribs.

thoracotomy in just such cases of encysted empyema as these—cases where repeated puncture failed to locate pus; where the physical signs were suggestive; and where the symptoms were urgent. Realizing that the patients would die of their empyema unless the pus were drained, it seemed better to me to run the risk of failure to relieve them by exploration, rather than to fold my surgical hands together in impotence, and reiterate to the physicians who called me as consultant, what I know they

[1] "Surgical Experiences with Encapsulated Empyema and Abscess of the Lung; a Plea for Exploratory Thoracotomy"; Medical and Surgical Reports of the Episcopal Hospital, Philadelphia, 1916, iv, 226.

had heard before in similar circumstances from other surgeons, a request to the physician to find the pus, with the statement that then, and then *only*, would the surgeon undertake to drain it! Some surgeons, I may add, are even more conservative than this, and even though pus has been found by puncture by the physician, refuse to undertake any operation unless they themselves succeed in finding pus by a puncture made on the operating table. To my mind, pus is pus, whether found by the physician or surgeon; and I believe if it is found within the pleura the chances that it will be absorbed and cause no further trouble are so very remote that

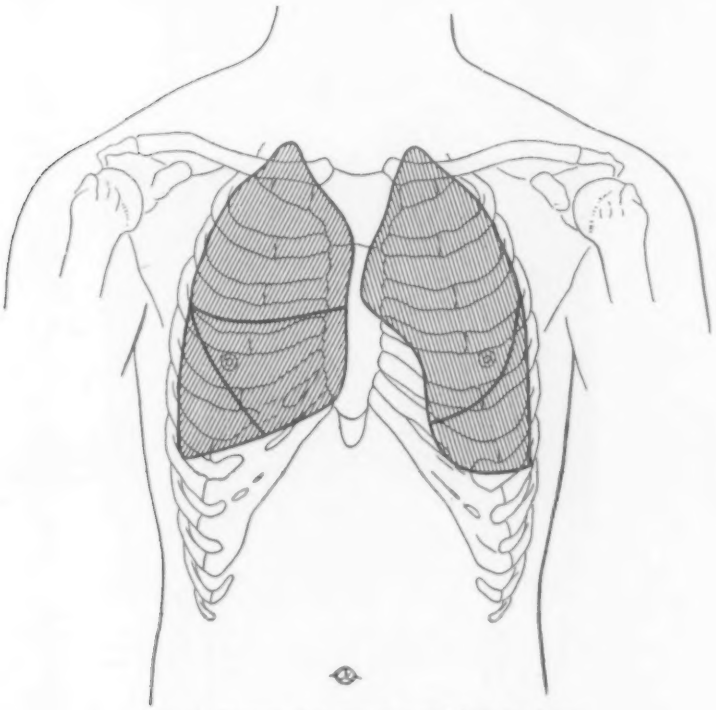


FIG. 4.—Fissures of the lungs viewed from the front.

unnecessary delay in resorting to operation is unjustifiable. In some cases, of course, it is possible that an undrained interlobar empyema will drain itself by rupture into a bronchus; but it has yet to be shown that this event improves the patient's chance of recovery. Of two cases in my series (Cases VIII and XIII) only one recovered.

In the report to which allusion has already been made, I recorded:

1. A case of empyema encapsulated between the upper and middle lobes of right lung, discharging through the bronchus; punctures negative; empyema found on exploratory operation; death from exhaustion and sepsis (Case VIII of the present series).

2. A case of empyema encysted between the upper and middle lobes of

the right lung. Numerous punctures negative; empyema not located at exploratory operation, but later burst into drainage tract; ultimate recovery (Case XVIII of the present series).

3. A case of empyema encysted between the left lung and the diaphragm. Punctures negative; empyema found on exploratory operation; recovery (Case XXIV).

4. A case of empyema encapsulated between the upper and middle lobes of the right lung. Puncture negative; empyema found on exploration; recovery (Case XXV).

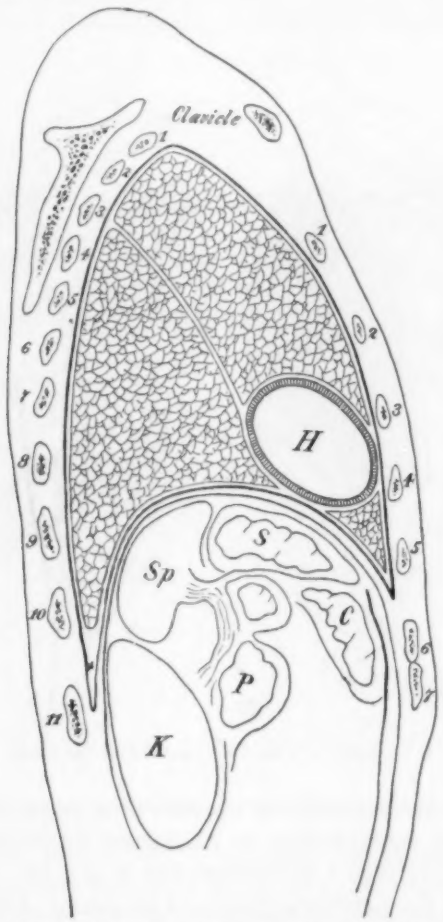


FIG. 5.—Sagittal section of thorax through middle of left clavicle.

5. A case of empyema encapsulated between the left lung and pericardium. Punctures negative; empyema not found at exploratory operation; death eight days later from continuing sepsis (Case XXVI).

Now I think it is worthy of note that in the last mentioned case, had an X-ray examination been available before operation, it is extremely

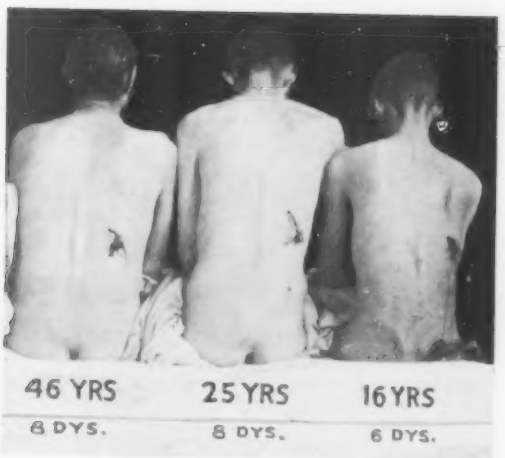


FIG. 6.—The site of election for draining an empyema; ninth, tenth or eleventh ribs posteriorly. The ages of the patients are given above, and the interval since operation below.

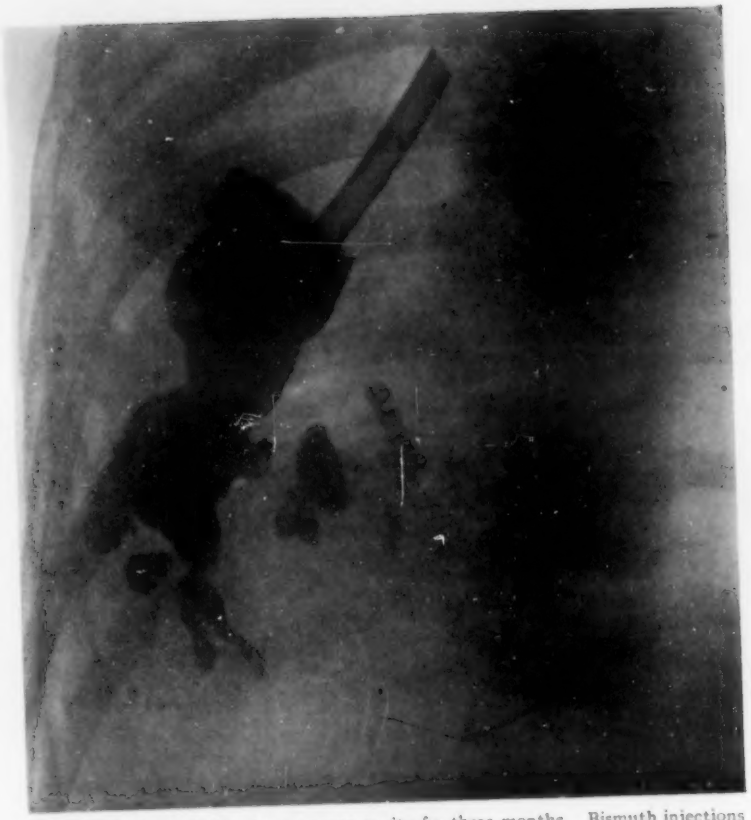


FIG. 8.—Rubber tube lost in empyema cavity for three months. Bismuth injections unavailing. Sinus healed permanently three weeks after removal of tube and Estlander operation.

FIG. 9.—Empyema cavity (*L.*) injected with bismuth paste, showing outlines of calcified pleura; ten months' duration. Died ten days after Estlander operation.

probable that the empyema might have been located when the chest was opened; since X-ray examination subsequent to operation (after necessary repairs to the X-ray plant) showed distinctly the outlines of the abscess cavity, and since autopsy showed that the interlobar dissection made at the time of the operation almost had reached the abscess.

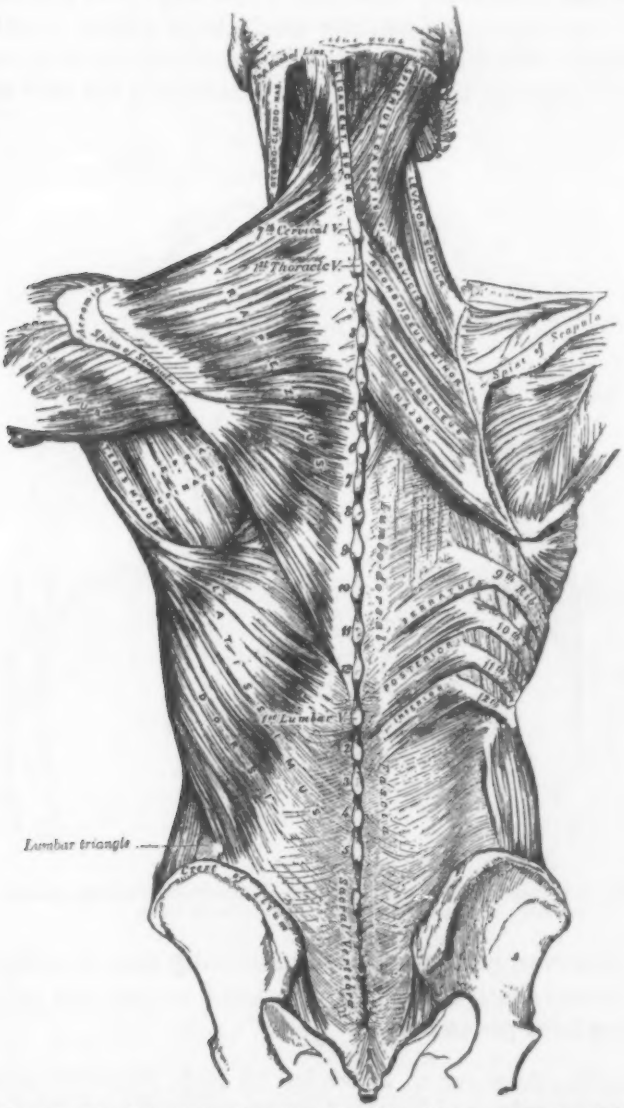


FIG. 7.—The ninth, tenth, eleventh and twelfth ribs exposed on the right side by removal of the latissimus dorsi. Note that the eighth and higher ribs are overlapped by the scapula. (From Gray's Anatomy.) Courtesy of Messrs Lea and Febinger.

So that, with regard to the *first and second propositions* laid down twenty-six years ago, I think we must understand them as permitting and encouraging at the present time, *exploratory thoracotomy,* when symp-

toms are urgent, medical measures have failed, repeated punctures are negative, and yet the presence of pus seems certain. Under these circumstances I have opened the thorax for exploration on six occasions (Cases VIII, XVIII, XXIV, XXV, XXVI, XXX), and except in the two cases just mentioned (Cases XVIII and XXVI) succeeded in finding and draining the empyema every time; and while only three of these patients recovered, I am sure more patients would have died if I had not been willing to explore the thorax; and if I had been allowed to do exploratory thoracotomy sooner in Case VIII, it is possible his life also might have been saved.

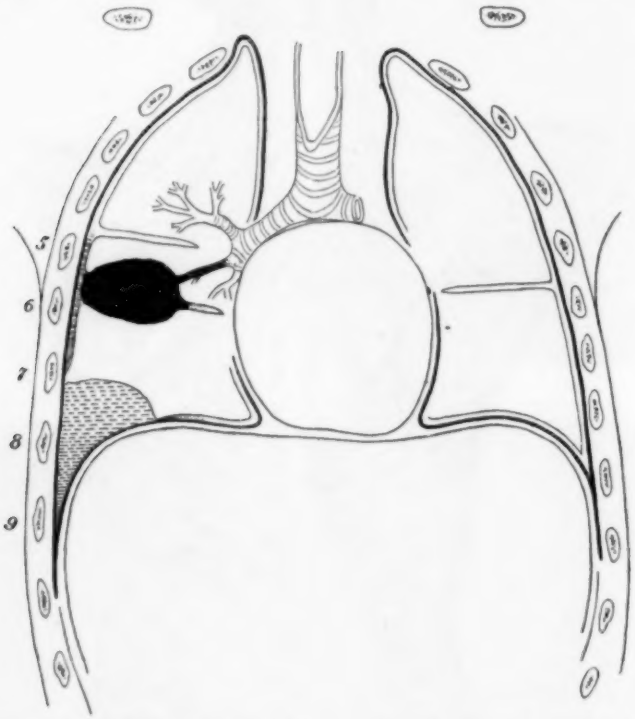


FIG. 10.—Case VIII. Interlobar empyema ruptured into a bronchus (pleural vomica). Note pleural effusion in costo-phrenic sinus.

I must, however, put on record the following case, in which I opened the thorax after negative puncture and found no pus, the patient subsequently dying from pneumonia.

Mildred C., three years. December 16, 1919: Admitted to the Episcopal Hospital, service of Doctor Carson, referred from Medical Dispensary by Dr. R. S. Hooker, with diagnosis of pneumonia. Has had a cold for three weeks with cough and expectoration; not confined to bed. Is fretful and unable to breathe properly at night. On admission: Temperature 101° F. (by rectum); anæmic, rachitic child, with Harrison's groove and rachitic rosary pronounced. Res-

pirations very short and rapid. Expansion of chest very asymmetrical. Apex beat of heart in midaxillary line about sixth interspace; marked pulsation over entire left chest. Rate rapid and sounds of good quality, no thrills or murmurs. Breath sounds rather harsh and many moist râles are heard over entire chest; no evidence of consolidation on percussion. Abdomen is distended, liver enlarged; no dullness in flanks. Extremities negative.

December 17th: Slight cough, no expectoration. Throat is clear. Not restless.

December 21st: Improving. Temperature 98° to 100° F.

December 30th: Temperature steady at 99° for last two days. Much improved. Sent home.

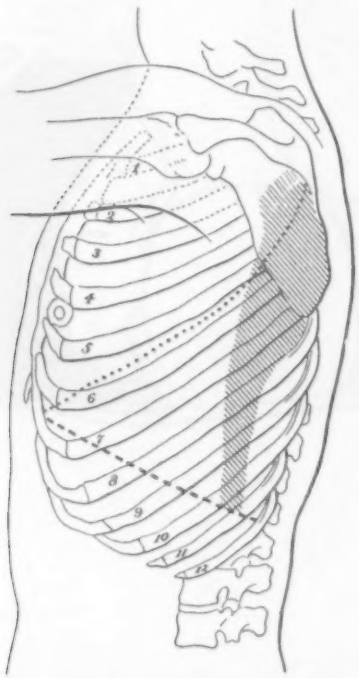


FIG. 11.—Case XV. Large, but distinctly encapsulated empyema.

January 6, 1920: Brought to Medical Dispensary and again seen by Doctor Hooker. Since being home the cough has continued and the child's general condition has been poor. Diagnosis of left-sided empyema was made by Doctor Hooker from dullness and diminished breath sounds. Sent into hospital and seen in Receiving Ward by Doctor Ashhurst.

Puncture of left chest over area of impaired resonance is negative. Under local anæsthesia 2 cm. of tenth left rib is resected, in mid-scapular line. Few adhesions between lung and diaphragm and between lobes of lung, but no pus found. Rubber tube inserted in

expectation that pus will later rupture into drainage tract. Time of operation twenty minutes.

January 7th: Condition poor. Lips cyanotic; respirations rapid and labored. No pus from tube. High-pitched bronchial breathing at left apex.

January 8th: Condition worse. Patient died at 5.15 P.M. Exploration of wound with finger after death could detect no adhesions between lobes and no evidence of empyema. Death from lobar pneumonia.

Technic of Exploratory Thoracotomy.—Let me here repeat what I understand by exploratory thoracotomy:

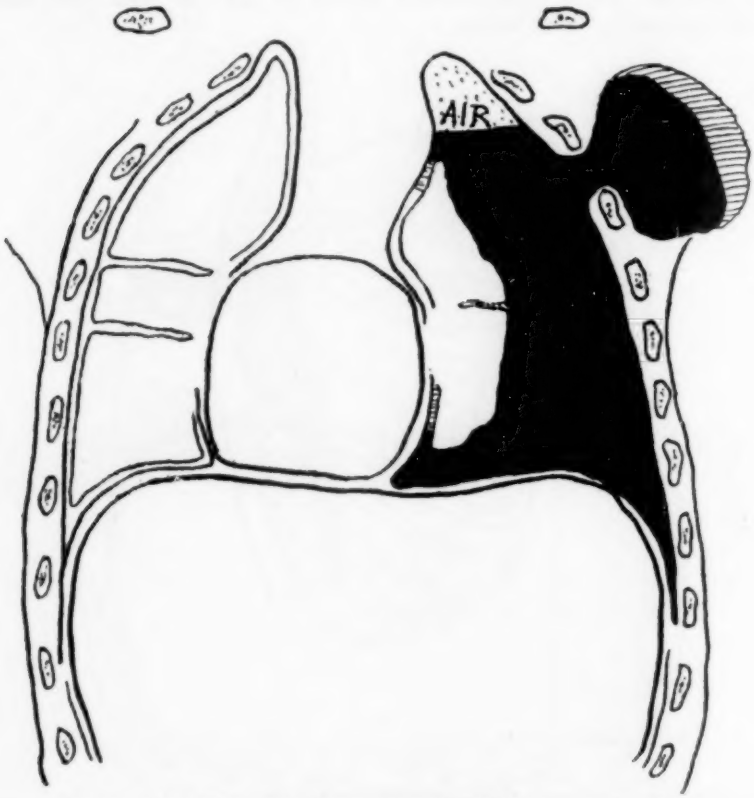


FIG. 12.—Case XVII. Pulsating empyema necessitatis = pyo-pneumo-thorax.

The patient is given, about thirty minutes before the commencement of the operation, a hypodermic injection of morphine (0.010 gm.) and atropin (0.00045 gm.); these doses may be larger in robust adults and smaller in children.

The patient is placed *prone* on the table, is made comfortable with pillows, and his head is turned away from the side on which operation is to be done. The arms are placed above the head. Care is necessary to see that the patient lies flat on his abdomen and thorax, and is *not turned on the healthy side,* as this will hinder respiration.

The proposed skin incision, over the eighth or ninth rib, back of the angle of the scapula, is then infiltrated with the local anæsthetic,[2] and the intercostal nerves above and below the rib to be resected are blocked with an injection of about 2 c.c. each of the anæsthetic fluid. These nerves are located by making the point of the needle impinge upon the rib above the nerve, and then prodding the rib with the point of the needle until the lower border of the rib is found, when, after pushing the needle about 0.5 cm. further into the intercostal structures the injection is made. This should be at a point 8 to 10 cm. from the spinous processes. When the anæsthetic has been properly injected the incision of the soft parts and the resection

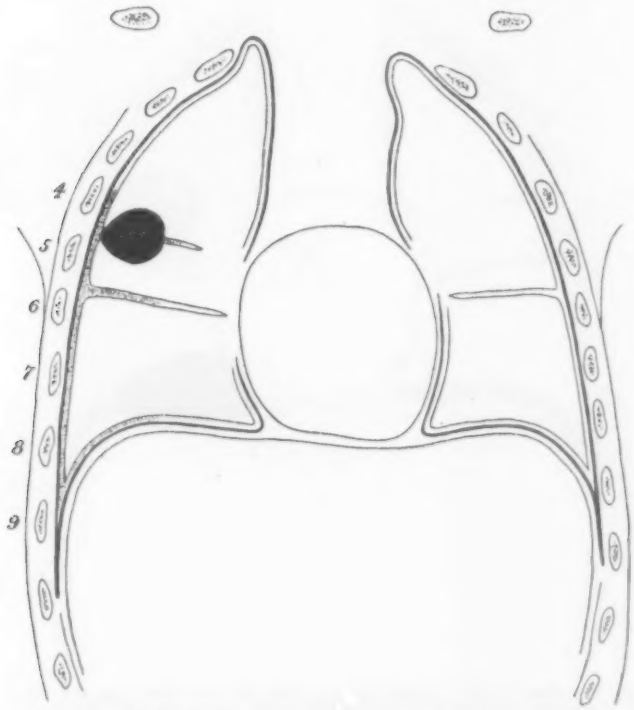


FIG. 13.—Case XVIII. Empyema encysted between upper and middle lobes of right lung; not found at operation. Later ruptured into drainage tract.

of the rib may be done absolutely painlessly. If, after the pleura has been opened and explored, more room is desired, it is perfectly easy to resect the rib next above or below by (1) infiltrating the skin parallel to the spinal column upward or downward from the posterior end of the primary incision; (2) blocking a third intercostal nerve as already indicated; (3) raising the soft parts in a flap; and (4) resecting (subperiosteally) as much as is required of the next rib. Usually it is not necessary

[2] I prefer novocain, 1:400; but when this was not available have used eucain, 1:100; to every 30 c.c. of the solution is added one drop of adrenalin chloride solution (1:10,000). This quantity (30 c.c.) usually is sufficient.

to divide the intercostal structures between the ribs resected, since when the ribs have been removed the intervening soft parts may be drawn aside by retractors. The length of rib to be resected depends on the size of the thorax and the extent of the intrathoracic exploration. As a rule, from 8 to 10 cm. is sufficient.

The pleura is opened between forceps, as the peritoneum is treated. Usually in cases of suspected empyema there is no collapse of the lung, which is adherent to the costal or diaphragmatic pleura. Even if the lung is not adherent it seldom collapses to less than half its bulk, when the

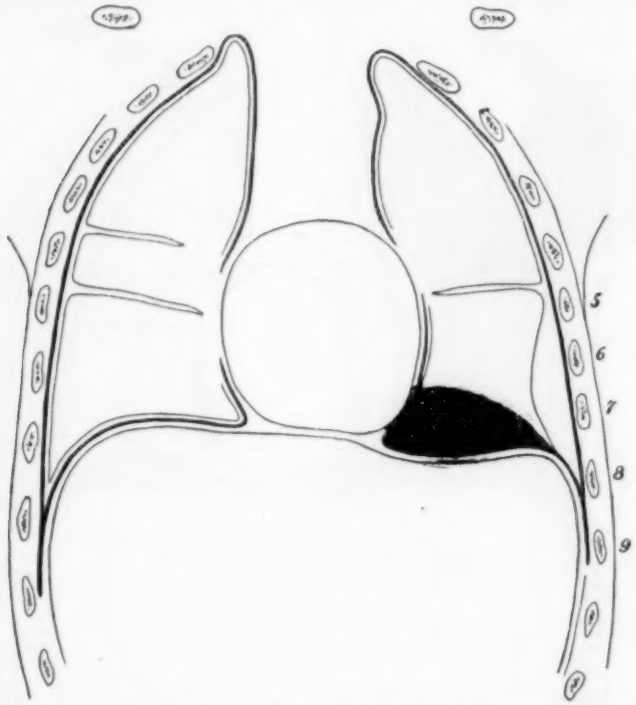


FIG. 14.—Case XXIV. Empyema encysted between left lung and diaphragm. Case XXXVI was quite similar, but on the right side.

patient is lying prone, and there is comparatively little respiratory disturbance. Wide opening of the pleura causes less dyspnœa than a small and valve-like opening which favors the development of a tension pneumothorax.

After opening the pleura and before conducting any exploration for an encysted empyema, the structures should be inspected. The lung may be so closely adherent to the diaphragm that it will be very difficult to recognize the line of junction. After ocular inspection, which may give a clue as to the first place to be searched for the abscess, the remainder of the pleural cavity should be walled off with hot moist gauze. Then with fingers, dissecting forceps, or even knife and scissors (according to the density of the adhesions), the lung should be gently released.

Usually it is well to clear the diaphragm first. If no pus is found here, the packs are rearranged so as to protect the lower part of the pleural cavity, and a search is begun in the fissures of the lung. If the lung is not readily accessible, its lower lobe is caught in two or three pairs of volsella forceps, and maintained within reach. A single pair of volsella forceps is more apt to tear out and so damage the lung than are two or three simultaneously applied. Fixing the lung in the wound steadies the mediastinum, enables the diaphragm to resume its piston action, and thus ventilates the other lung. The lung is absolutely insensitive to these

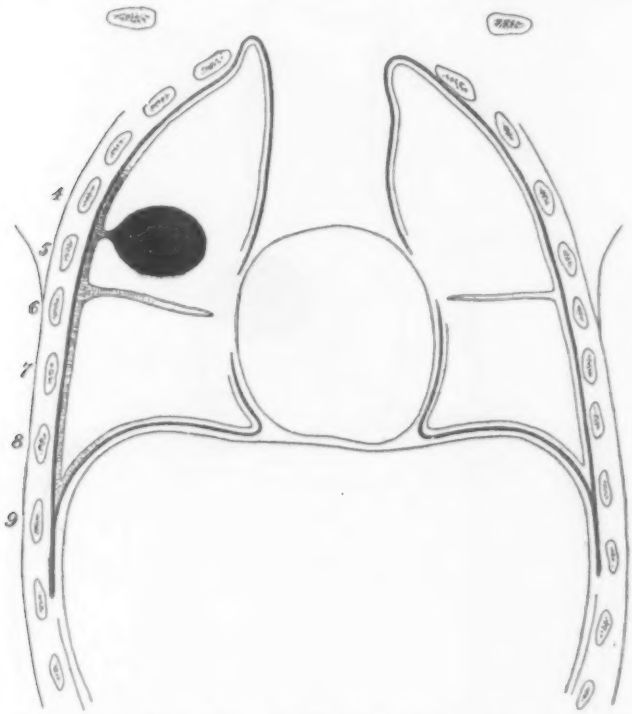


FIG. 15.—Case XXV. Empyema encapsulated between upper and middle lobes of right lung. Evacuated by thoracotomy. Compare Fig. 13 (Case XVIII).

manipulations. Often it is not necessary to fix the lung in this way, and the surgeon proceeds at once to separate the lobes from each other. Usually where the adhesions are densest, pus will be found.

When pus is finally located, and has been evacuated, a rubber tube (with a lumen at least of 1 cm.) surrounded with loose iodoform gauze is placed in the abscess cavity, the isolating packs are removed, and the wound is closed in layers, but not too tightly, around the drainage tract—the pleura and intercostal structures in one layer, then the skin.

The frequency of encapsulated empyema I am sure is not recognized. In my experience it has been present in no less than one-fourth of the cases. It is true, of course, that every empyema is, in a sense, encapsu-

lated; in other words, that it does not involve the entire pleural cavity. Pleuritis is in a way analogous to peritonitis: only so long as the effused fluid is sero-pus is it unconfined by adhesions. By the time frank pus has formed, in every case, whether in the peritoneum or the pleura, adhesions set certain limits, large or circumscribed, to the cavity in which pus is found. The only "free pus" that can exist in a body cavity such as the pleura or peritoneum is pus which is suddenly effused in overwhelming quantities into previously normal pleura or peritoneum, by the rupture of a localized abscess (appendicular or pelvic abscess, empyema of

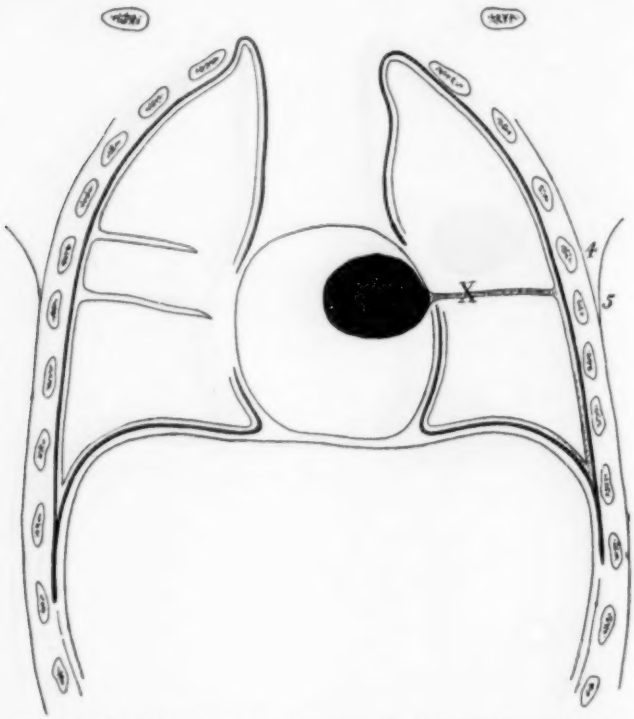


FIG. 16.—Case XXVI. Empyema encapsulated between fissure of left lung and pericardium. X indicates the point reached at operation.

the gall-bladder, abscess of the liver or spleen, encysted empyema, etc.). But what I understand by encapsulated empyema is either one which comes into contact with the costal pleura at no point (Cases XVIII, XXIV, XXV, XXVI, XXVII, XXXVI) or over so small an area that it is difficult, if not impossible, to locate pus until after the thorax has been opened (Cases VII, VIII, XXVIII, XXIX, XXXV), or until an empyema necessitatis has formed. It should be recognized also that there may be more than one distinctly encapsulated empyema in the same patient (Cases XVI, XXIX, XXXV); and that occasionally an hour-glass empyema may exist, the channel of communication being large (as in Cases XXXVII and XLIII), or so small that drainage of one loculus is ineffective in producing cure of the other (as in Case XXXVIII). It is on

account of the occurrence of cases such as these that I urge that, if the condition of the patient permits, exploration should be adequate to ensure that no such encapsulated foci are overlooked. (I am not prepared, however, to adopt Lilienthal's "Major thoracotomy" under general anæsthesia, so long as I continue to find a medium thoracotomy under local anæsthesia satisfactory.) In all cases with massive empyema, of course,

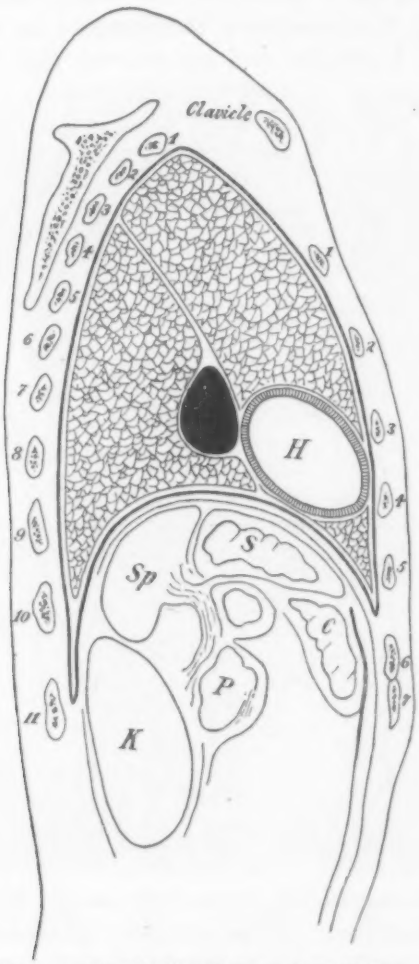


FIG. 17.—Case XXVI. Diagram of sagittal section through middle of left clavicle, indicating site at which pus was found at autopsy.

removal of a large amount of the pus by aspiration should precede by one or two days the formal operation of thoracotomy; and in no case where the fluid can be removed by aspiration need drainage by thoracotomy be regarded as an emergency operation (*third proposition*).

Let me revert now to the *fourth proposition* I have quoted at the beginning of this discussion, namely, "that drainage is best effected by making two openings, one at the lowest available point, and carrying a

large drainage tube through the cavity from one opening to the other." It seems to me that the doctrine of through-and-through drainage merely follows as a corollary in cases where the primary opening has been made too high; in other words, that if the primary opening is made at the site of election, which I regard as the ninth or tenth (occasionally the eleventh or twelfth) ribs near their angles (Figs. 6 and 7); it is unnecessary to make a counter-opening, as one is already at the "lowest available point." I repeat, that in cases of exploratory thoracotomy as well as in cases of massive empyema, I practise and recommend that the opening should

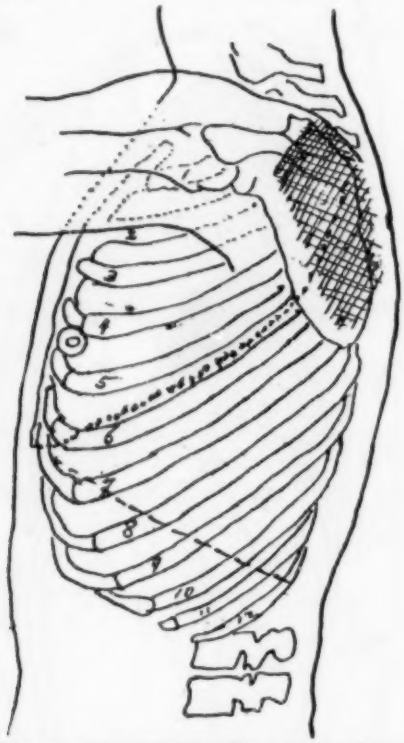


FIG. 19.—Case XXVIII. Empyema encapsulated over posterior end of left interlobar fissure.

be made at the site mentioned, even if the needle has shown pus in the sixth or seventh interspace. It is so nearly invariably the rule that the empyema cavity extends down to the diaphragm, and it is so easy after opening the pleura low down to drain an empyema which may extend already to within a few centimetres of the opening, by breaking through its limiting adhesions at their most dependent portion; and a higher opening unless supplemented by a counter-opening at the lowest available point gives rise to so very prolonged a convalescence, that I believe there are exceedingly few cases in which it is desirable to be limited in selecting the site for drainage by the location at which pus was found on puncture. Only once since I adopted this rule have I encountered an empyema

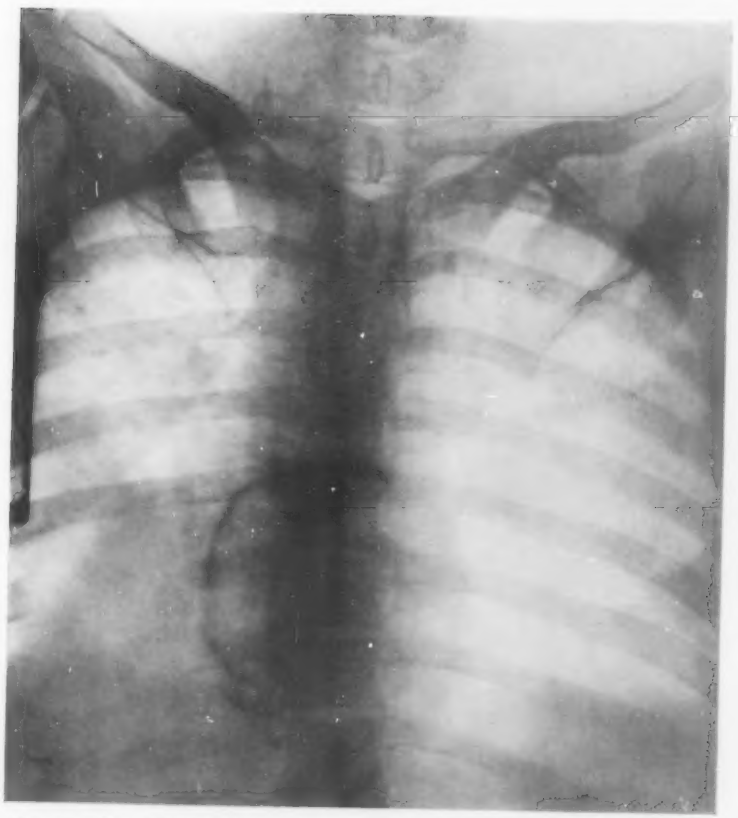


FIG. 18.—Case XXVI. Encysted empyema three days after operation, at which no pus was found. Note distinct outlines of abscess cavity overlying shadows of sixth, seventh and eighth thoracic vertebræ, mostly to left of midline.

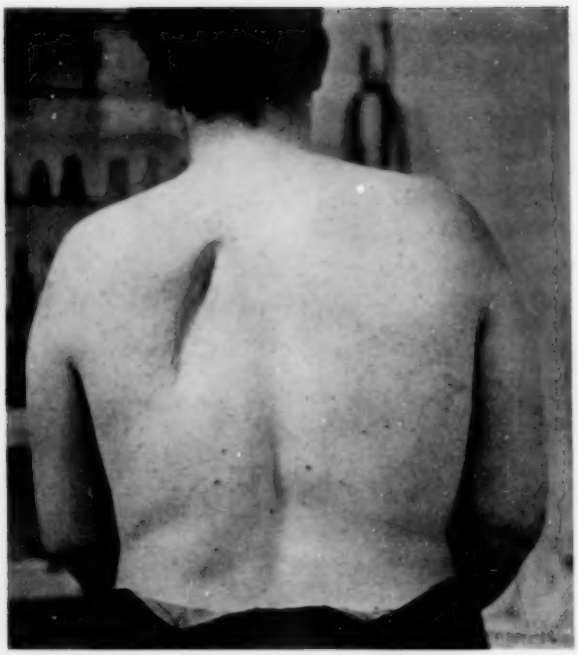


FIG. 20.—Case XXVIII. Resection of fourth and fifth ribs. Three years and a half after operation.

situated so very high and with the lower portion of the thorax so certainly intact, as to induce me to make a higher opening: in this patient (Case XXVIII) the empyema appears to have formed between the lobes of the left lung and to have finally come into contact with the parietal pleura between the vertebral column and the scapula at the level of the spine of the latter bone; and yet the drainage tract at this site, though it was nearly at the lowest level of the empyema cavity, nevertheless required over four months for healing.

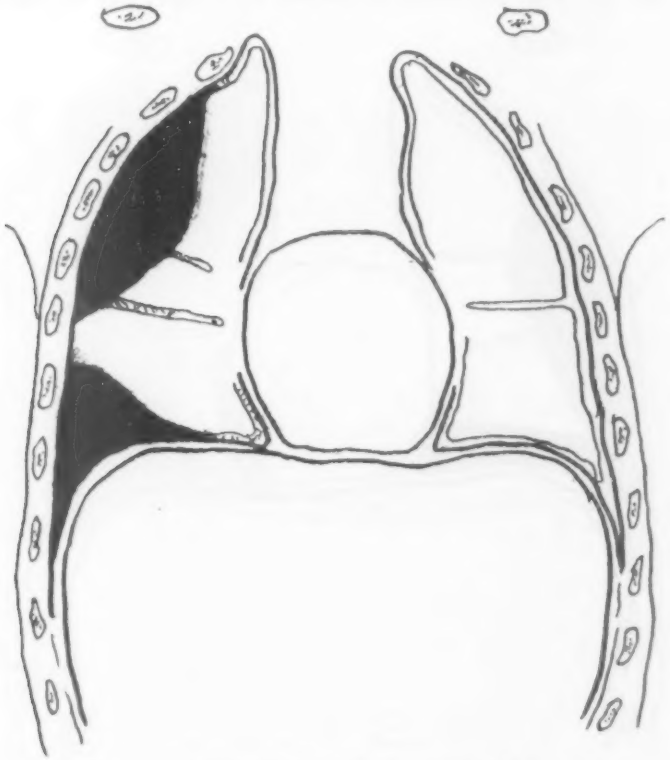


FIG. 21.—Case XXXV. Two distinct encapsulated empyemata.

Resection of a rib I regard as important in all cases. Intercostal incision alone does not afford adequate drainage, and frequently secondary operations are required. I have employed it only once (Case IV) in a baby six months old; a month later it was necessary to resect a rib to improve the drainage, but two months after the first operation the patient died with unhealed sinus, probably from sepsis arising in the inefficiently drained pleura. In another case which came later under my care (Case XIII), intercostal incision was employed by another surgeon, but this healed upon an infected cavity, and the empyema not being drained externally ruptured into a bronchus; finally, the original operative sinus opened again, and eventually I had to resect the eighth and ninth ribs;

definitive healing then occurred in eight weeks. I have never seen necrosis of the rib ends, and cannot believe the possibility of such an occurrence is to be regarded as contra-indicating rib resection.

Next we come to the *fifth proposition*, "that drainage should be supplemented by washing out the cavity with mild antiseptic fluids. When the lung has expanded and discharge has nearly ceased the tube should be shortened . . . and gradually withdrawn." With the very gradual shortening and withdrawal of the tube I am in entire accord, but I do not see that anything is gained by irrigations in the ordinary

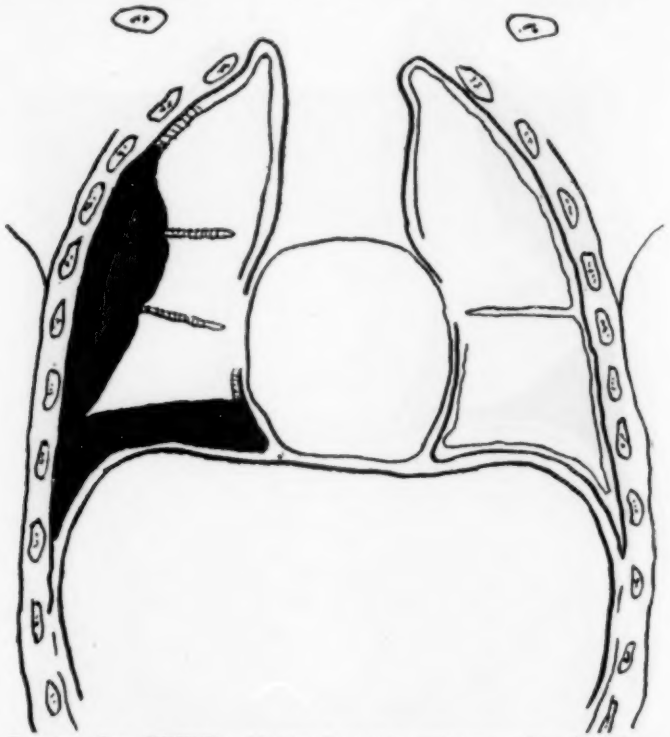


FIG. 22.—Case XXXVII. Massive hour-glass empyema. Case XLIII was quite similar.

case of empyema. I suspect that the main function they served in my father's day was to keep the lumen of the through-and-through drainage tube open, and that the fluid injected at one end of the tube almost immediately escaped by the other end, without making a very extensive journey into the empyema cavity. And I am quite sure that in some cases injudicious irrigations are productive of harm. It is quite evident, I believe, that death in two of my patients (Cases XXX and XXXI) some weeks after they passed from under my care, is directly attributable to sepsis arising in the wound in the thoracic wall, not in the empyema cavity, and that this sepsis was produced by irrigation, and probably aided in Case XXXI by too tight closure of the wound of the second operation.

That irrigations with Dakin's hypochlorite solution, by the Carrel technic, are of no value in any cases, I am not prepared to admit. But I am quite thoroughly convinced that in the vast majority of cases of empyema they are quite unnecessary, and that except in cases of very long standing, with much thickened pleura and non-expansile lung, healing will occur quite as promptly, as pleasantly, and as definitively by means of large and dependent drainage. I do not think it can be denied that in many patients of the class of cases just mentioned the irrigations with

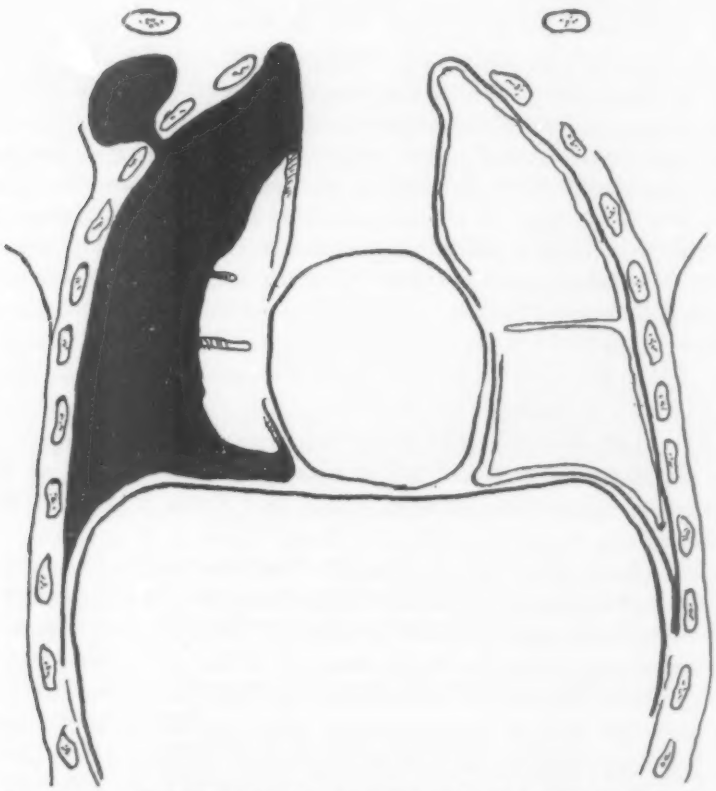


FIG. 23.—Case XXXVIII. Massive empyema necessitatis, pointing beneath right pectoral muscles. With metastatic abscess between left scapula and chest wall.

Dakin's solution will eat away the inflammatory lymph deposited on the visceral pleura and that the lung will thereafter expand sooner and more completely than if such irrigations had not been employed. Nor can it be denied that it is possible to sterilize an empyema cavity by this means, at least temporarily, and then to permit the thoracic wound to close upon the sterile pneumothorax; but I also know that the same event may take place simply as the result of efficient drainage without any irrigations, at any time (Cases XIV and XXXV). Speaking for myself, I may say that I have never employed the Carrel-Dakin treatment for empyema,

and that I should be surprised to encounter a recent case in which I thought it indicated.

The *sixth proposition,* which has to do with unhealed empyema cavities, I am sure must undergo great modification in view of advances in surgery since it was formulated. I am convinced that unhealed empyema is infrequent compared to its incidence a generation ago, largely due to the earlier recognition and more efficient treatment of the acute cases. Indeed, it is probably true that if any surgeon conscientiously follows up the treatment the occurrence of an unhealed case will be extremely rare. One patient (Case VI) on whom I operated in 1909 is known to have had his sinus still unhealed nine months after operation; but among the remainder of my patients the longest period of healing was seven months, and in not one of those who remained under my care was a second operation required. One patient (Case XXXI) who passed from my care two weeks after the operation, and from whom the tube was removed too early, was reoperated on later for an accumulation of pus and eventually died of sepsis arising in the thoracic wound of the second operation. Another patient (Case XXXV) who was tuberculous and whose thoracic wound healed within seven weeks upon a pneumothorax, five months later had a little discharge from the sinus which healed again in three weeks; twenty-two months after my operation another empyema developed and was drained; seven months after this second operation the patient moved to Colorado, and since that time (September, 1919) he has had one slight reaccumulation which was opened and healed at the end of six weeks. He writes (March, 1920) that he is now apparently in perfect health.

I should like to say a good word for bismuth injections in the treatment of unhealed sinuses, as advocated by Ochsner. In one patient (Case XXXVI) (the only one in whom I began to despair of seeing the sinus close spontaneously) and in whom removal of the tube was followed on several occasions by rise of temperature, I had just about determined on reopening the wound (twelve weeks after operation) and injected it with 10 per cent. bismuth paste to ascertain the outlines of the cavity by skiagraphy: the next day the sinus was found healed, with the paste retained, and the wound remained healed when the patient was last seen five months later.

Unhealed empyema must be rare in Philadelphia, as I have had occasion to operate on not more than two patients.

In the *first patient,* John K., sixteen years of age, operation had been done by another surgeon April 19, 1911, for a left-sided empyema five weeks after the onset of pneumonia. On June 11th the tube was not found when the wound was dressed, and on June 17th the patient was discharged with unhealed sinus. Soon after this he came under my care in the Episcopal Hospital, and was treated with bismuth injections. The sinus sometimes would close for a few days but

always reopened. Finally, an X-ray picture was taken and showed the tube lying in the pleural cavity (Fig. 8).

September 15, 1911 (three months after the first operation): Under ether, I resected about 6 cm. each of the seventh, eighth, and ninth ribs (the eighth, which had been excised at the former operation, was of cartilaginous consistency). The drainage tube and large masses of bismuth paste were removed, and a fair amount of pus was evacuated. All the lymph adherent to the visceral pleura that could be detached was removed, all the adhesions of the lung to the parietal pleura were broken through, and the cavity thoroughly wiped with iodoform gauze. One long and wide strip of iodoform gauze was left in for drainage.

In two weeks the wound was practically closed, and was firmly healed by October 7, 1911, twenty-two days after operation.

In the *second patient*, James K., fifty-one years of age, there was a history of some severe injury to the chest twenty-eight years previously. He had had many attacks of "pleurisy" following occasional attacks of rheumatism.

May 24, 1913: Admitted to the Episcopal Hospital, Doctor Mutschler's service. About fifteen months previously he had developed a heavy cough followed by swelling in left thorax, diagnosed by family physician as tumor. The family physician incised this swelling and quite a large quantity of pus was removed. The sinus healed up after a long time. There had been no discharge for the past four or five months. About ten days ago he began coughing again and the sinus opened and discharged pus. Has been having night sweats. Does not spit any blood.

On admission: Sinus in left thorax between sixth and seventh ribs, in nipple line, from which a small amount of pus is oozing. Two scars of healed sinuses. Temperature 101°. Large-framed man, thorax well developed, expansion limited on left which is dull front and back below fourth rib, with distant breath sounds and harsh crepitant râles.

May 26, 1913: X-ray 6442—heart displaced to right by pus in left pleura up to sixth rib.

May 27th: Operation by Doctor Mutschler (ether): Sixth left rib excised in anterior axillary line and much pus discharged (pneumococci).

May 30th: Temperature normal.

June 28th: Still free discharge. Temperature hectic since last note. 98°–101°. X-ray 6640—heart still to right, some collapse of left lung.

July 25th: Still same amount of discharge. Temperature 98°–100°.

August 1st: Service taken over by Doctor Ashhurst. The patient walks about and does not complain of anything; general condition about same. Dressings always saturated over night. Coughs a little.

August 8th: X-ray 7213 (bismuth) shows large cavity extending from eleventh rib to upper thorax (Fig. 9).

August 23d: Discharge no less. Temperature 98°–100°.

August 29th: Operation—ether (Ashhurst). Patient prone. Incision 12 cm. long made over sinus of previous operation (over sixth left rib in anterior axillary line), and about 10 cm. each of the sixth, seventh, eighth, and ninth ribs resected, from their angles forward. This uncovered very dense parietal pleura, which was found to be lined by a calcareous deposit about 5 mm. thick, as were also the exposed surfaces of the lung and diaphragm. The rigid walled cavity extended as high as the finger could reach through the wound. The calcareous lining was peeled off the parietal pleura, the diaphragm, and the lung, except at the apex of the cavity where a few plates had to be left, as it was impossible to remove them without tearing the lung. The lung was found still to be bound down by dense pleura, beneath the calcareous deposit, but the condition of the patient did not warrant continuing the operation by decortication or discission of the pleura. The lung moved a little more in respiration than before the calcareous plates were removed, but it was evident that only by collapse of the soft parts could the cavity be obliterated; and though complete obliteration was not hoped for, it was thought the marked reduction in size of the cavity would considerably reduce the discharge and promote the patient's comfort. Drainage was provided by iodoform gauze and a large rubber tube. The wound was closed in layers about the drainage.

August 31st: Dressed, dressings soiled but not saturated, as before operation. No calcareous matter could be felt by finger introduced through wound.

September 6th: Temperature fell after last dressing to normal and has not varied more than one-half degree since. To-day rose to 100°. Seems irrational, no cough.

September 7th: Restless, on gradual decline. Pulse weak, rapid (150). Temperature 101°. Respiration 34.

September 8th: Died 6 A.M. Temperature having risen rapidly to 104°. No post-mortem, but by opening wound calcareous matter found only around apex of cavity. Lung and diaphragm covered with gray lymph, phlegmonous in type.

It is a question in my mind whether in this latter patient the condition of his pleura did not date from his original injury twenty-eight years previously. Certainly it would be very unusual for such dense calcareous deposits to have occurred between the time of the two operations done in the Episcopal Hospital only three months apart; but the presence of a hemorrhagic pleural effusion many years ago, which remained unabsorbed, will account for the patient's repeated attacks of pleurisy as well as for the densely calcareous deposit. In this connection the appearance of the X-ray in Case XXVI (Fig. 17) is suggestive that here also a cavity containing sterile fluid may have persisted for over four years, this patient having been under treatment in another hospital for supposed empyema, which could not be located, that length of time before she came under my care with recurrence of similar symptoms.

CONCLUSIONS

From this discussion it seems to me that the propositions laid down twenty-six years ago must be modified in the following particulars:

1. Cases of pleural effusion suspected of being purulent should be punctured (with hollow needle attached to air-tight syringe); and if the effusion is massive most of it should be removed by aspiration one or two days before thoracotomy is undertaken.

2. If the fluid found on puncture is serous, or sero-purulent, thoracotomy usually may be postponed, until frank pus has formed, as this delay will permit the formation of firmer adhesions and thus prevent complete collapse of the lung when the empyema is opened. Cures of such sero-purulent effusions, however, have so rarely occurred without final resort to thoracotomy that attempts to cure them by injection of antiseptic fluids into the unopened pleura are usually detrimental to the patient.

3. If in a case of suspected empyema the symptoms are urgent, but pus cannot be found by puncture, exploratory thoracotomy should be undertaken in an effort to locate and drain the pus.

4. The operation of thoracotomy for empyema should provide free and dependent drainage, secured by resection of a rib, usually the ninth, tenth, or eleventh, in front of its angle. This operation may be done with perfect satisfaction to both patient and surgeon *under local anæsthesia,* and in most cases this is preferable to a general anæsthetic. (I have used local anæsthesia with satisfaction in 27 patients, including one baby sixteen months of age; and have employed it in every one of my last 20 patients, some of them requiring rather extensive intrathoracic manipulations. It is interesting and perhaps significant to note that only two patients in the entire series (Cases III and XI) developed pneumonia of the opposite lung after operation, and that both these patients had taken ether.)

5. Post-operative irrigations are unnecessary, unless after several months the lung shows no tendency to expand; when the use of Dakin's fluid may prove beneficial. In selected cases (those with small cavity) injections of bismuth paste may procure closure of the sinus.

6. If the cavity cannot be made to heal by these means, the surgeon should do a major thoracotomy, combined with decortication of the lung and discission of the pleura, and in some cases resection of a number of ribs to permit the chest wall to collapse in part and meet the expanding lung.

In closing, I may be permitted to give a summary of my own cases, few in number, perhaps, but studied with such attention as was possible. Among 42 patients there have been 9 deaths, a general mortality of 20.9 per cent. Among the first 9 of these patients there were 5 deaths, three of which were in babies less than a year of age. But if there are included only the last 34 patients operated on since adopting the system of wide and de-

pendent drainage,[3] there were in this number only 4 deaths, a mortality of less than 12 per cent. Moreover, of these four fatal cases:

(*a*) In the first (Case XXII) the child also had extensive gangrenous stomatitis (noma) and died from this.

(*b*) In the second (Case XXVI) the empyema (which was encapsulated against the pericardium) was not found at operation, and the patient died eight days after operation unrelieved.

(*c, d*) In the third and fourth cases (Cases XXX and XXXI) death occurred some weeks after the patients passed from my care, and as the result not of their empyema, but from sepsis arising in the thoracic wound and brought on by irrigations.

From which facts I conclude that the mortality of empyema, if promptly and efficiently treated, need not exceed 10 per cent.

The *average time required for firm healing of the thoracic wound,* in 24 cases in which this is known, was just over nine weeks, the shortest being three and the longest thirty weeks. In those patients (18 in number) who remained under my personal supervision, the average time until firm healing was just over seven weeks, the shortest time being three and the longest twelve weeks.

In 5 cases (Cases XXX, XXXI, XXXVI, XXXIX and XL) (2 of these patients (Cases XXXVI and XXXIX) being still under my personal care at the time) there was more or less interruption to the progress of wound healing by pocketing of pus, due to too early withdrawal of drainage; and in 2 of my early cases (Cases IV and V) where dependent drainage was not secured at the first operation, secondary operation was required some time after they passed from my care. With the exception of Case V, a patient who passed from under my care after the operation, there were no cases unhealed at last note, and in none had an Estlander or similar operation been required to secure closure of the empyema cavity.

Certain cases in the subjoined list deserve more than passing attention.

Cases of encapsulated empyema, with limited contact with parietal pleura (Cases VII, VIII, IX, XV, XVI, XXI, XXVIII).

Cases of encapsulated empyema, without any contact with parietal pleura (Cases XVIII, XXIV, XXV, XXVI, XXVII, XXXVI).

Cases of two distinctly encapsulated empyemata within same pleural cavity (Cases XVI, XXIX, XXXV).

Cases of hour-glass empyema (Cases XXXVII, XXXVIII).

Cases of empyema necessitatis, Cases VI, XVII (pulsating), XXI, XXXVIII.

Cases of empyema as part of a general septicæmia, Cases XIV (peritonitis and abscess), XXIV (streptococci in blood), XXXIII (jaundiced), XXXIV (pericarditis, endocarditis), XXXVIII (metastatic abscess and pyæmia).

[3] It is proper that I should give due credit to T. Turner Thomas, whose studies in 1913 turned my attention particularly toward the value of dependent drainage.

Cases of encapsulated empyema rupturing through a bronchus, Cases VIII, XIII.

Cases of tuberculous empyema, Cases XII, XVII, XXXV.

Case in which drainage tube fell into empyema cavity, Case XVI.

Case complicated by noma (gangrenous stomatitis), Case XXII.

Case complicated by independent pleural effusion on same side, Case XXI.

The subjoined table gives the salient features in all the cases encountered, and, in accordance with the motto "*Ars medica tota est in observationibus*" abstracts of the case histories are appended.

CASE REPORTS

CASE I.—Empyema, right. Recovery. John C., aged five years.

September 22, 1906, admitted to the Children's Hospital, Dr. Hutchinson's service. Ill for six weeks with pneumonia at Atlantic City; then in Children's Hospital (Dr. Charles H. Weber's service) for the last three weeks. Empyema suspected, but Doctor Weber had four dry taps. Last night pus was found (diagnosed as interlobar in position by Doctor Weber).

September 22, 1906: Operation (Doctor Ashhurst)—ether. Excised 2.5 cm. right eighth rib in mid-axillary line. Pleura thickened (50 to 60 mm.); incised, and incision dilated with hæmostat. Pus oozed out with little force, then as child began to cry on coming out of ether (ammonia substituted for ether after opening pleura), pus was ejected with much force, creamy, pale yellow, sour. Pus appeared to extend from diaphragm up, not interlobar (only). Before operation respirations were 60, fell to 40 immediately after and to 22–24 after two hours. Pus gave a pure culture of pneumococci.

November 8, 1906: Tube out.

July 27, 1907: Seen at Country Branch of Children's Hospital, in excellent health; wound closed.

CASE II.—Empyema, left. Died, one week (enteritis). Joseph S., five months.

July 26, 1907: Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Had been ill three weeks with pneumonia. Doctor Hand aspirated (July 25, 1907) about 50 c.c. of pus (diplococcus, biscuit-shaped).

July 26, 1907: Operation (Doctor Ashhurst)—ether. Resected 2.5 cm. of eighth left rib, about 100 c.c. thick inodorous pus. Time—six minutes. Child very ill. Temperature normal after operation and did well for four or five days, then got severe enteritis, with high fever. Lungs clear, wound clean.

August 2, 1907: Died, one week after operation, from enteritis.

CASE III.—Empyema, left. Died, two weeks (pneumonia of other lung). Edward W., aged eleven and a half months.

July 25, 1908: Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Has been in hospital about two weeks with pneumonia; temperature came to normal, but rose again on July 22d and July 23d. Doctor Hand aspirated July 24th and got 25 c.c. pus.

Examination.—No dyspnœa lying down; lessened expansion, dulness, absence of breath sounds at left base. Mucous râles at apex.

July 25, 1908: Operation (Doctor Ashhurst)—ether. Excised 2 cm. rib (about eighth) below angle of scapula, patient on back. About 100 c.c. thick creamy pus, slight odor (staphylococci and few diplococci). Rubber tube.

July 28, 1908: Since operation almost no discharge of pus.

July 29, 1908: Temperature up, cyanosis, dull over lower part upper lobe.

July 30, 1908: Doctor Hand suggested collection of pus.

July 31, 1908: Sinus explored with finger, only firm adhesions found, lung expands well. No pus by needle inserted in the dull area. Bronchial breathing above empyema cavity.

August 4, 1908: Pneumonia in right lung discovered.

August 8, 1908: Died of pneumonia of right lung.

CASE IV.—Empyema, left. Died, two months (sepsis from pleura?). Katharine P., aged six months.

July 22, 1909: Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Pneumonia four weeks ago. Aspirated (Doctor Ashhurst) and 100 c.c. pus (pneumococci) evacuated. Did well until July 24, when dyspnœa returned.

July 24, 1909: Operation (Doctor Ashhurst)—ether. Intercostal (seventh to eighth?) incision, left; about 500 c.c. of pus evacuated, and tube inserted.

July 27, 1909: Doing well.

August 2, 1909: Has had rapid respiration, 72–84; pulse, 160–170. Temperature, 101° to 102° for last few days. Wound doing well. Eats well; bowels normal.

August 4, 1909: After taking ammonium carbonate, syrup of senega, respirations are only 60–70. Pulse 140–150. Temperature 99°. Still miserable.

August 14, 1909: Much improved.

August 19, 1909: No more improvement.

Later in August, Doctor Hodge resected a rib to secure better drainage. In September the child was taken home, taking a milk mixture. Death towards the end of September, two months after operation, perhaps from sepsis from the pleura, the wound not having healed.

CASE V.—Empyema, left. Recovery. Joseph T., aged six years.

July 24, 1909: Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Had pneumonia five weeks ago. Aspirated ten days ago at home, one litre of pus obtained. On admission, the apex beat of heart is to right of sternum. Marked orthopnœa. After aspiration of 100 c.c. no more pus would run.

Operation (Doctor Ashhurst)—ether; resection of 2 cm. of eighth rib in left anterior axillary line, and evacuation of about a litre of pus and flocculent lymph.

July 27, 1909: Pulse still weak, no fever. Lying down for first time since operation. Bloody pus discharging.

July 29, 1909: Tube removed, only blood-stained serum discharged and smaller tube replaced.

August 3, 1909: Tube left out; nothing in sinus. Slight discharge.

September, 1909: Doctor Hodge resected another rib, for better drainage. Doing well in end of month.

March 9, 1910: Readmitted and record shows only use of bismuth paste. Discharged April 23, 1910. "Discharge disappearing rapidly." Cannot be traced.

CASE VI.—Empyema, left. Recovery. Elias R., aged three years.

July 29, 1909: Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Pneumonia five weeks ago. Developed deformity of chest which family physician called "post-pneumonic pigeon-breast." On admission had empyema necessitatis (left), apex beat in second interspace right. Whole left thorax tense, with fluid between ribs and skin and muscles. Aspirated by resident, large quantity of creamy pus.

July 29, 1909: Operation (Doctor Ashhurst)—ether. Resected 2 cm. of sixth or seventh rib in anterior axillary line and evacuated over a pint or less creamy pus.

July 30, 1909: Sitting up and playing around bed.

August 4, 1909: Uneventful recovery after this date.

CASE VII.—Empyema, left. Recovery. Sam S., aged five years.

July 22, 1909: Admitted to the Children's Hospital, Doctor Hand's service. Illness began July 18th with chill and fever. Has had temperature of 103° since. Pneumonia

in left lung. Empyema suspected yesterday, but two punctures by Doctor Hand drew no pus. Transferred to Doctor Hodge's service July 29th.

July 29, 1909: Doctor Ashhurst punctured twice in anterior axillary line, left, where auscultation and percussion indicated pus, but found nothing. Then punctured in seventh interspace in posterior axillary line, where was most tenderness and very slight œdema, but no auscultatory signs, and drew very foul pus from considerable depth. Expectoration slight but very foul, like pulmonary abscess (mixed growth).

Operation.—Ether. Resected 3 cm. of eighth left rib in posterior axillary line, without injuring pleura, suspected pulmonary abscess, but on incising pleura quantity of thin turbid pus (colon smell) squirted; later some coagula of lymph. Drainage tube.

July 30, 1909: Temperature normal, little discharge.

August 5, 1909: Temperature normal ever since operation.

August 25, 1909: Temperature up for one or two days, two weeks ago, and again yesterday and to-day. Uneventful recovery therafter.

Case VIII.—Empyema, right, discharging through bronchus. Died third day. Pleural vomica. Sepsis. William L., aged fifty-two years.

July 26, 1912: Admitted to the Episcopal Hospital; under Dr. E. J. Morris's care until August 1, 1912, then on Doctor Robertson's service.

Family History.—Father, grandmother and brother died of tuberculosis.

Past History.—Pertussis as child. Tobacco and beer to excess.

Present Illness.—Began ten days ago as sudden sharp pain in right side, after lying on that side on grass for an evening. Pain gradually subsided after few days. Then got short of breath. Family physician gave medicine without effect. Walked to dispensary and sent to ward.

Examination on Admission.—Large, full-blooded, dyspnœic; constant cough and expectoration. Breath very fœtid. Right chest: flat below fourth rib, with hyperresonance above. Left apex: impaired resonance. Râles and harsh breathing at right apex and below fourth rib. Absent breath sounds and fremitus. Expectorates 500 c.c. daily of very foul smelling pus. Temperature, 99°–100°.

July 27, 1912: Constant cough. Cannot sleep, cannot speak above a whisper. White blood count, 16,680. No tubercle bacilli found in sputum in five separate examinations; elastic tissue absent.

August 6, 1912: Seen by Doctor Eves for aphonia; examination negative except for acute laryngitis.

August 18, 1912: Growing weaker. Temperature ranges from 99° to 102° F.

September 3, 1912: Aspiration negative on right, below angle of scapula (Doctor Robertson). Still septic.

September 7, 1912: Tapped by Doctor Robertson (negative) below angle of right scapula. Oxygen introduced into pleura until dyspnœa was produced and entire area of dulness became tympanitic. Dyspnœa soon disappeared and patient became comfortable.

September 9, 1912: Dulness as before over right chest.

September 10, 1912: Oxygen again introduced by Doctor Robertson. Patient nearly died, was unconscious for fifteen minutes; pulse 150.

September 11, 1912: Improved, dyspnœa disappeared. Temperature 99°–103° F.

September 13, 1912: Transferred to Doctor Frazier's surgical service. Seen by Doctor Ashhurst, who thought it to be an encapsulated empyema rupturing through bronchus. Patient is very dyspnœic and coughs frequently until expectoration of very foul greenish material. Pulse fairly strong, 92–96. Respiration, 28–32. Temperature, 100°; no pain. Whole right chest is flat from middle of scapula down; breath sounds weak, many crackling râles. Breath sounds amphoric below angle of scapula for distance of two ribs. Repeated punctures have located no pus.

September 14, 1912: Operation (Doctor Ashhurst)—eucain 2 per cent. in intercostal nerves of eighth and ninth ribs, and by infiltration also. Incision in eighth interspace

posterior axillary line, patient lying on face. Excised 5 cm. of ninth rib and 6 cm. of eighth rib. Ligated both intercostals front and back. Patient complained of pain *in abdomen*. This exposed diaphragm. Bloody serum evacuated on opening pleural cavity; lung stripped easily from ribs except beneath scapula (sixth to eighth ribs, in front of angles). Here it was densely adherent. Packed remainder of pleural space. Lung densely adherent to dome of diaphragm and to posterior parietal pleura beneath scapula. Burrowed with finger among these adhesions beneath scapula, and ruptured into abscess, which discharged with a gush 30 c.c. or more of same pus as is expectorated. (Aromatic ammonia to stifle fœtid stench.) This cavity was beneath sixth and seventh ribs in posterior axillary line, about 7 by 5 cm.; though finger could not reach upper limits (Fig. 10). Inserted rubber tube (1.25 cm. in diameter) and iodoform gauze around tube. Time, one-half hour. Patient asked for his supper as soon as he was turned over on his back, and was given it on return to ward.

September 15, 1912: Very dyspnœic, pulse very rapid and weak. Tube to lung drains large amount of greenish pus. Dressed wound.

September 16, 1912. Died 4:30 P.M.

CASE IX.—Empyema (encysted), left. Died, six days (sepsis). Minnie Y., aged fifty-nine years.

December 10, 1912: Admitted to Episcopal Hospital, Doctor Piersol's service. Ill for four days, with pain in left chest. Examination showed pneumonia of left lower lobe.

December 15, 1912: Temperature fell by crisis (ninth day).

December 20, 1912: Empyema suspected—dulness persists, impaired fremitus. Very ill.

December 24, 1912: Temperature suddenly rose to 104.3°. More toxic. Tapped, thick greenish pus from left pleura, base of lung (culture, pneumococcus).

December 25, 1912: Transferred to Doctor Frazier's surgical service. Patient appears to be insane. Very emaciated and weak, bed-sore, etc.

Operation (Doctor Ashhurst).—Cocain, 2 per cent. Patient prone. Injected intercostal nerves at and below eleventh rib at angle. Then waited five minutes. Skin not anæsthetized yet, so infiltrated it (15 minims of 2 per cent. cocain). Same amount had been used for each of two nerves). Then entire operation (resection of one and a half inches of eleventh rib) was done absolutely painlessly. After removing rib, found pleura unruptured. Opened this (as peritoneum) with scalpel, and found lung lightly adherent, and showing no tendency to collapse. Packed pleural cavity off above with one strip of gauze, then burrowed in region of densest adhesions (just anterior to opening) and thick (semi-solid) pus welled up with each respiration. Inserted one-half inch rubber tube (about two and a half inches inside thorax), packed it around with iodoform gauze; withdrew isolating gauze; dressed wound, and turned her on her back.

December 27, 1912: Doing well. Temperature fell to normal after operation and remained there..

December 29, 1912: Temperature 96°.

December 30, 1912: Temperature 103°. Lungs negative. Drains well. Bed-sore slough removed.

December 31, 1912: Died from exhaustion and sepsis from bed sore.

CASE X.—Empyema, left. Recovery. Mary O'B., aged five years.

January 10, 1913: Admitted to the Episcopal Hospital, Dr. Piersol's service. Ill three days with pneumonia of left lower lobe. Temperature reached normal by lysis on January 17, 1913.

January 20, 1913: Temperature 101°–104°. Aspiration in ninth left interspace gave a large amount of pus (diplococcus pneumococcus).

January 22, 1913: Transferred to surgical service. Temperature still high.

Operation (Doctor Ashhurst).—Ether. Patient prone. Resected 3 cm. of ninth rib, in front of its angle; gush of yellow curdy pus (about 250 c.c.). Drain. Rubber tube.

February 18, 1913: Tube discontinued.

March 10, 1913: Out of bed.

March 21, 1913: Discharged. Wound healed.

September 28, 1916: Social Service Department reports; has moved and cannot be found.

CASE XI.—Empyema, left. Recovered. James F., aged seven years.

February 10, 1913: Admitted to Episcopal Hospital, Doctor Piersol's service. Ill three weeks at home, feverish and coughing. Effusion in left lower chest. Needle gave thick, greenish pus. Culture, pure pneumococci. Temperature, 102°. Delirious.

February 12, 1913: Temperature normal. Flat all over left chest, posteriorly.

February 14, 1913: Operation (Doctor Ashhurst)—ether. Patient prone. Excised 3 cm. of eleventh rib at angle. Gush of pus when periosteum was torn through, before fragment of rib was excised. No hæmostats required. About 500 c.c. of yellow inodorous creamy pus, with large amounts of lymph. Lung not much bound down by adhesions, and expanded well after removal of pus. Drain tube 1 cm. lumen, inserted about 5 cm., so as to be above level of diaphragm when this rose.

February 17, 1913: Pneumonia at right apex. Delirious, very weak. Temperature since operation, 99°–102° F.

February 21, 1913: Temperature normal.

February 27, 1913: Bed-sore over sacrum.

March 11, 1913: Slowly improving.

March 21, 1913: Tube replaced.

April 3, 1913: Bed-sore healed.

April 11, 1913: Tube removed.

April 20, 1913: Tube replaced.

April 28, 1913: Tube removed. Out of bed.

May 10, 1913: Discharged. Sinus gives slight discharge.

September 13, 1916: Healed three or four weeks after leaving hospital. No deformity; rib has reformed. Impaired resonance and a little distant breath sounds. Well and hearty.

CASE XII.—Empyema, left. Recovered. Walter H., aged fifteen years.

August 21, 1913: Admitted to Episcopal Hospital, Doctor Frazier's service.

Family History.—No tuberculosis.

Past History.—Pneumonia at five years; well since. School-boy.

Present Illness.—In October, 1912, noticed some swelling on left side, dull pain. Broke open in one month and discharged a little pus. Dressed twice daily since. Slight cough, frequent night sweats. Very little expectoration.

Examination.—Right apex impaired resonance. Left is fair. Right apex bronchial breathing and pectoriloquy. After each five or six beats of heart a very loud friction rub is heard, evidently pericardial, and systolic in time. From sixth rib to base on left is dull front and back. Scars of two healed sinuses, anteriorly in seventh interspace. Sinus below angle of scapula discharges whitish inodorous pus. Probe enters four inches and detects bare carious rib.

August 23, 1913: White blood count, 18,320.

August 26, 1913: Operation (Doctor Ashhurst)—eucain, 2 per cent. One and a half inches of tenth left rib removed; great amount of greenish pus exuded. (Smear: few micrococci; culture: negative.) Tube.

August 28, 1913: Not much pus.

September 6, 1913: Discharge less, irrigated daily.

September 8, 1913: Blows Wolfe bottles.

September 12, 1913: Better expansion of left.

September 20, 1913: No discharge. Sent home. Temperature never above 99° after operation.

September 28, 1916: Three years after operation reported to have left the city.

CASE XIII.—Empyema, left. Recovered. Edna P., seven years.

June 25, 1913: Admitted to Episcopal Hospital, Doctor Mutschler's service. Had been ill for six weeks. Acute onset with meningeal symptoms. In bed since with pain in left side. Weakness and cough and yellowish expectoration. Whole left chest full of fluid.

June 27, 1913: Operation by Doctor Mutschler. Intercostal incision, sixth to seventh ribs, left; large amount pus evacuated.

July 1, 1913: Improved. Temperature irregular but not high.

July 20, 1913: Only small sinus remains.

July 29, 1913: Temperature rising.

August 1, 1913: Service transferred to Doctor Ashhurst. Temperature, 103°. Signs of fluid; two taps negative. Old sinus entirely healed.

August 2, 1913: White blood count, 26,000.

August 5, 1913: Temperature, 100°–102°. X-ray.

August 10, 1913: Coughed up some pus; sent to Laboratory. Negative for tubercle bacilli; diplococcus predominates.

August 13, 1913: Sinus ruptured during night. Temperature lower.

August 19 to August 24, 1913: No improvement. Patient feels ill. Temperature up.

August 25, 1913: Better; thick, yellowish expectoration. X-ray after bismuth injection shows cavity extending from fourth to eleventh ribs.

August 26, 1913: Temperature normal.

August 27, 1913: Operation (Doctor Ashhurst)—ether. Patient prone. Excised 3 cm. of ninth and eighth ribs, near angle, opening cavity in pleura (no communication with bronchi apparent); removed bismuth; broke up some adhesions. Drain tube and iodoform gauze. Large amount of bloody pus removed.

September 18, 1913: Tube removed.

October 22, 1913: Entirely healed, out of bed.

October 27, 1913: Discharged.

September 6, 1916: Very slight scoliosis convex to left. (Left ribs were resected.) Ribs reformed. No disability. Attends State Dispensary for Tuberculosis.

February 21, 1920: Nearly seven years since operation; patient now fourteen years of age. Grandfather, living in same house, died of phthisis soon after her return home from hospital. Examination shows expansion of chest limited at both apices, but the lungs are normal. There is no increase in the slight left convex scoliosis noted three and a half years ago, and no disability.

CASE XIV.—Empyema, left (pleuro-peritonitis). Recovered. Margaret P., aged twelve years.

October 27, 1914: Admitted to the Episcopal Hospital on Doctor Fussell's service, Had been ailing off and on for three weeks before onset of acute illness, but attended school daily. One week before admission had sudden violent pain in lower left abdomen. Began vomiting and has been vomiting ever since. Castor oil not effectual. Bowels not open for one week. No cough. Pains her to be moved and to take deep breath. Pains to urinate or make any motion with abdominal muscles.

Examination on admission by Dr. J. W. Moore: Temperature, 103°; pulse, 136–120. Well developed and well nourished, but anæmic. Lips red, face pale. Breathing rapid, but rather full respiration. No œdema or cyanosis. Not apathetic. Teeth good. Tonsils very large and red and pharynx red. White blood count, 30,000; polynuclears, 75 per cent.; hæmoglobin, 75 per cent.; red blood count, 4,220,000.

Chest: Expansion good and equal on both sides. No thrills or frictions. Tactile fremitus normal everywhere. Whole lung resonant to percussion except an area, size

of quarter, under angle right scapula, which is dull, and an area in right midclavicular line fourth rib (anteriorly), which seems hyperresonant, almost tympanitic. Breath sounds vesicular *all over*, including dull area, but here is heard an occasional crackling râle. No bronchophony or whispered pectoriloquy.

Heart: Negative.

Abdomen: Very rigid and tender all over, especially in epigastrium, right and left iliac fossæ. Not scaphoid nor markedly distended. Spleen and liver not palpable because of rigidity, but spleen not enlarged to percussion and liver dulness present. Abdomen resonant throughout, no dulness in flanks. Active peristalsis all over abdomen.

Rectum: No masses or areas of exquisite tenderness.

October 28, 1914: Examination by Doctor Fussell.—Patient looks ill. Complains of pain in abdomen when moved. There is a red flush on right forehead. Constantly makes outcries because she states she has pain in her stomach. Slight crusted scar on lower right lip, may or may not be herpes. Slight glandular enlargement in both axillæ and both groins. Breathing rapid and rather quick. No dulness on left side in front or laterally. Dulness on right, beginning at fifth rib in mid-axilla and merging into liver dulness. No dulness on right side behind. No blowing breathing posteriorly on either side. During examination dulness in right axilla disappears. Liver dulness begins at fourth rib parasternal line and extends almost to edge of the ribs. Splenic dulness is apparently increased. In respiration the abdominal wall is absolutely fixed. On palpation there are no masses or any particularly tender points. There is a great deal of abdominal tenderness in addition to the rigidity. Halfway between xiphoid and umbilicus the respiratory murmurs are curiously well heard. There is no sign of peristalsis. (Treatment just after admission is not recorded.) Rectum is ballooned out, but there is no mass.

Transferred to Doctor Ashhurst's service (day after admission): Temperature 103°; pulse, 120–130; respiration, 38–40; white blood count, 31,000. Eyes bright, cheeks red, rapid costal breathing. Abdomen very rigid all over, a little distended below umbilicus. Three doctors had examined her by rectum and felt no mass. Doctor Ashhurst did not examine chest or rectum; he thought it a surgical case (peritonitis), but that child was too sick for operation. Transferred to surgical ward and enteroclysis given, *nothing* by mouth (still vomiting frequently, and has been having water in medical ward, but no enteroclysis) and continued head-high posture. Also camphor and digitalis hypodermically (not given in medical ward).

October 29, 1914: Got out of bed last night and managed to get a little water (delirious). White blood count, 23,840; polynuclears, 92 per cent. (Nothing by mouth, continued enteroclysis. Fowler position.) Abdomen distended, tympanitic to percussion everywhere, save in right iliac fossa, where a dull area can be made out. Right side more rigid than left. Has vomited only twice. Nose-bleed at 3.30 A.M.

October 30, 1914: Much weaker and more restless. Abdomen not so distended, but boardlike rigidity over entire abdomen. Does not vomit, has not passed any flatus; paristalsis can be heard in upper abdomen; complains of great thirst. Still nothing by mouth, and enteroclysis. Temperature, 101°–103°. Pulse, 120; respiration, 26–30. Given digitalis and strychnin. No sign of localization of peritonitis.

October 31, 1914 (Saturday): Decidedly better. Upper abdomen just a little softer. Suspicious baggy feel by rectum (Doctor Ashhurst's first examination). Respiration still very rapid. Temperature, 99°. Pulse, 110–115. By noon, hot water given by mouth. By afternoon (Doctor Ashhurst's third visit this day), feels almost convalescent, she says. Complained of pain "around heart" and left lower chest at A.M. visit; P.M. temperature, 101°.

November 1, 1914 (Sunday): Temperature, 101° (steady); pulse, 108–120; respiration, 32–40. Respirations still very rapid. Left cheek flushed. Alæ nasi play violently. Abdomen rather rigid, no dulness, rectal examination negative. Chest

negative anteriorly. Doctor Ashhurst determined on thorough chest examination (his first), because of failure of abdominal abscess to appear. No evidence of subphrenic abscess (Hoover's sign). Right lung negative. Left dull from angle of scapula down, posteriorly and in axillary line, not anteriorly. Tender on pressure in intercostal spaces here. *Perhaps* slight bulging of these spaces. Breath sounds distant. *Needle* inserted in seventh interspace below angle of scapula drew pus (diplococci in chains). Doctor Fussell came in consultation and said subphrenic abscess, *no* evidence of empyema. Doctor Ashhurst maintained it was ***empyema thoracis.***

Operation (Doctor Ashhurst).—Gas, 2.30 P.M. Resected eighth rib in anterior axillary line; 150 c.c. pus from pleura (streptococci). Tube. Liquids by mouth. At 10 P.M. abdomen less tender, lungs œdematous. Heart good. Given morphine and atropine. Temperature, 104°. Pulse, 120–140. Respiration, 40–48.

November 2, 1914: Profuse discharge of pus from pleura; heart displaced to right. Abdomen softer, normal bowel movement (first since enema October 29). White blood count, 47,760; polynuclears, 89 per cent.

November 3, 1914: Temperature falling. Continue morphin, atropin and stimulation. Three normal bowel movements (no purge).

November 4, 1914: Better, abdomen softer, no bowel movement.

November 5, 1914: Temperature gradually falling. Pulse, 120–130. Respiration, 30–38. Bowels open daily from now on. Examination by Doctor Fussell. Labored breathing. Heart: right border one and a half inches to right of sternum, sounds are very rapid and feeble. Right chest anteriorly and posteriorly many râles. Left chest: pneumothorax. Abdomen is tender, especially over McBurney's point.

November 6, 1914: Examination by Doctor Ashhurst. No rigidity of abdomen, no tenderness to a reasonable degree of pressure over appendix or any other part of abdomen. To all intents and purposes the abdomen is normal. Respirations are thoracic. Fair expansion on both sides of upper chest; below nipples expansion better on right than left. No breath sounds heard anteriorly on left; normal on right. Right chest posteriorly is normal, on left posteriorly—pneumothorax.

November 7, 1914: Herpes on lip and tongue.

November 8, 1914: Improved.

November 10, 1914: Profuse discharge from chest still.

November 14, 1914: Fluid suspected at right base, no puncture.

November 16, 1914: Tube shortened.

November 22, 1914: Tube removed. Heart in normal position. Left thumb opened for infection (pure staphylococcus).

December 5, 1914: Out of bed. Thumb badly infected. Furunculosis of back.

December 11, 1914: Convalescent for two weeks and in chair now and chest normal. Thin.

December 14, 1914: Chest healed.

December 17, 1914: Gained three pounds in last three days.

December 23, 1914: Severe cough. Examination by Doctor Piersol: Compensatory emphysema of upper and middle right lobes; slight fluid or thickened pleura at right base from seventh dorsal spine down. Consolidation or compression of upper left lobe; below effusion and air.

December 24, 1914: Feels fine. Examination by Doctor Ashhurst; dulness at right base, breath sounds distant, consolidation at left base; sounds diminished, pneumothorax. Abdomen negative.

December 25, 1914: This afternoon had attack of acute pain in abdomen, vomited once. Temperature, 100°. Abdomen tender throughout and some rigidity.

December 26, 1914: Temperature 99°–101°. Right lower quadrant rigid. Liquid diet.

December 30, 1914: Sequestrum removed from thumb.

December 31, 1914: Chest unchanged. Aspiration on right: no fluid.

January 3, 1915: Temperature, 99°–101°. Pulse, 104–120. Respiration, 28. Vomited this A.M.

January 4, 1915: No better. Slight pneumothorax anteriorly on left; empyema scar solid.

January 5, 1915: Examination by Doctor Frazier (rectal and abdominal), who found no abscess. Resident physician (Sproul) could not feel anything by rectum. Doctor Ashhurst examined patient again and found all abdomen below navel rigid and tender and dull, and in rectum a distinct bulging abscess. White blood count, 26,000 for last week. Polynuclears five days ago, 82 per cent.; yesterday, 72 per cent.

Diagnosis.—Pelvic abscess from appendix, relighted by Xmas dinner.

Operation.—Gas, 11:30 A.M. Incision right paramedian two inches, suprapubic. Transversalis fascia and peritoneum matted together and dense; at once on opening peritoneum white creamy pus welled out, about 50 c.c. in all. Finger inserted felt intestines matted together by adhesions; and burrowed to Douglas's pouch; rubber tube, and iodoform gauze. No sutures. Time, ten minutes. (Culture, streptococcus.)

January 6, 1915: Temperature normal; free discharge of pus; much better.

January 10, 1915: Bloody purulent expectoration. Sitting in chair.

January 13, 1915: Tube removed from abdomen.

January 15, 1915: Weight, fifty-four and a half pounds.

January 17, 1915: Slight discharge. Gaining weight.

January 23, 1915: Went home, weight fifty-nine pounds. Sinus closed.

October 4, 1915: Scar two inches. No hernia. No disability. Appendix has never been removed.

April, 1920. Family report patient in good health. Is a professional dancer.

CASE XV.—Empyema, right. Recovered. Hugh McI., aged forty-three years.

November 7, 1914: Admitted to the Episcopal Hospital to Doctor Frazier's service. Ailing all summer. Pneumonia four weeks ago, crisis three weeks ago, but did not get well, had fever, cough and marked expectoration. In bed all the time. Bed-sore beginning. Temperature, 101° F.; right lung from middle down gives absent breath sounds and fremitus, and is flat. Aspiration gives pus in seventh interspace posteriorly (pneumococcus).

November 8, 1915: Operation—eucain, 2 per cent. Patient prone. Resection of 3 cm. of ninth right rib in posterior axillary line. The sinus of empyema runs up posteriorly to the region of the third rib, where the cavity is larger. The lung is bound down tight by adhesions (Fig. 11).

November 9, 1914: Temperature normal.

November 18, 1914: Tube out.

November 23, 1914: Discharged.

September 28, 1915. Refuses to return for examination. Wife says he is in good health but has a cough.

CASE XVI.—Empyema, left. Recovered. Joseph R., aged nineteen years.

December 3, 1914: Admitted to Doctor Frazier's service, Episcopal Hospital. Pneumonia (left) three weeks ago; crisis two weeks ago, but did not get well. Fever developed with pain in left chest and dyspnœa. Cough and expectoration continued. Needle in seventh interspace shows pus. (Family physician.)

Examination on Admission.—Left base, absent breath sounds and fremitus with flatness. Hoover's sign positive. White blood count, 15,840. Temperature 103° F.

December 4, 1914: Operation—eucain, 2 per cent. Excised 2.5 cm. of eighth left rib posteriorly; 500 c.c. of pus (diplococci).

December 8, 1914: Tube lost in wound (was sutured to skin by No. 1 chromic gut). X-ray 3376a shows it inside chest.

December 11, 1914: Efforts yesterday failing to extract tube. Doctor Ashhurst to-day (gas) resected 2.5 cm. more off same rib at anterior and then at posterior end of former section, and finally found tube in spinal gutter posteriorly. *Large pus*

pocket opened and drained. Tube fastened with silkworm gut and safety pin. Temperature has been normal since operation last week.

December 14, 1914: Drainage has about stopped.

December 18, 1914: Out of bed.

December 22, 1914: Discharged.

September 28, 1916: Has moved to Wilkes-Barre, Pa.

CASE XVII.—Pulsating empyema necessitatis, left. Recovered. Gerald L., aged twenty-eight years.

December 10, 1914: Admitted to Doctor Frazier's service, Episcopal Hospital.

Past History.—Is tuberculous; has been ill off and on for seven years. In February, 1911, had left pneumonia, followed by pleural effusion (empyema); tapped, tube inserted (no rib resected). Acute illness lasted six months. Sinus ran nearly one and a half years. Recovered and resumed work about one and a half years ago (laborer).

Present Illness.—Ailing for one month, very ill for one week, high fever, bad cough, pain in chest; expectoration offensive and profuse.

Examination.—Extremely ill, delirious, cyanosed and dyspnœic. Empyema necessitatis over second left rib and in upper axilla. Former pulsates synchronously with heart. Above middle of scapula there is Skodaic resonance; below this flat, immobile (Hoover's sign very pronounced), breath sounds and fremitus absent. Over apex (L) breath sounds loud and blowing, almost amphoric. Right lung full of crepitant râles. White blood count, 50,000. Polynuclears, 91.3 per cent. Temperature, 99°. Pulse, 100–128.

Diagnosis.—Pyopneumothorax. Heart displaced 4 cm. to right of sternum. Thirty-eight ounces (well over one litre) of pus removed by aspiration in left eighth interspace posteriorly. Foul and fæcal smelling. (Long pale bacillus with streptococcus predominating.) After aspiration bulging area disappeared below clavicle, and this was sucked in with each respiration. Given morphin for cough (Fig. 12).

December 11, 1914: Still in poor condition, but much better than on admission.

Operation.—Eucain, 2 per cent. Excised 3¼ cm. of ninth rib (left). Patient lying prone. Pleura very thick (5 mm.) and prune juice looking pus evacuated after breaking up some adhesions. Exceedingly bad stink of H_2S gas. Long tube inserted.

December 12, 1914: Better; marked discharge. Temperature, 102°.

December 14, 1914: Temperature again normal.

December 18, 1914: Tube removed.

December 29, 1914: Went home against advice, but in very good condition, out of bed several days.

September 28, 1916: Has moved to Souderton, Pa.

CASE XVIII.—Empyema, right (encysted). Recovered. Tony V., two years.

November 10, 1915: Admitted to Doctor Frazier's service at the Episcopal Hospital. Three weeks ago became ill and treated at home for pneumonia; seen to-day by Dr. R. S. Hooker at home and diagnosed as empyema. On admission, temperature, 102°; pulse, 108–148; respiration, 32–40; white blood count, 25,000. Poorly nourished, thin profuse nasal discharge. Pharynx unusually red. Thorax: resonance good, a few râles low in right axilla, and breath sounds are harsh around angle of both scapulæ.

November 11, 1915: Aspiration (numerous punctures) of right pleural cavity, no fluid obtained. Temperature down.

November 13, 1915: Temperature up.

November 15, 1915: Aspirated again in left axilla, no fluid obtained.

November 16, 1915: White blood count, 36,640. Lymphocytes, 40 per cent. Polynuclears, 46 per cent. Hæmoglobin, 59 per cent.

November 17, 1915: Temperature down.

November 18, 1915: Temperature up.

November 19 to 23, 1915: Temperature, 100°–103°, irregular. Seen in consultation

by Dr. W. E. Robertson, who diagnosed fluid under the sixth rib right. X-ray confirms this.

November 23, 1915: Operation (Doctor Ashhurst)—ether. Time, ten minutes. Patient prone; resection of 3 cm. of seventh right rib near angle of scapula. Pleura thick. Opened this, no pus. Finger introduced found lung lightly adherent to parietal pleura and diaphragm all over. No dense adhesions. No interlobar collection between middle and lower lobes found. Only blood. Culture, sterile. Drain, rubber tube.

November 26, 1915: Wound doing well.

November 29, 1915: Temperature has fallen gradually to normal.

December 1, 1915: Sudden profuse discharge of pus through wound. (Culture, streptococci and staphylococci.) Evidently an encysted empyema had broken into wound (Fig. 13). Temperature, 101° F.

December 3, 1919: Considerable drainage.

December 8, 1915: Discharge scanty.

December 12, 1915: Chest still draining but scanty.

February 5, 1915: Went home. Sinus healed dry.

September 6, 1916: No deformity, lung normal, rib reformed.

CASE XIX.—Empyema, right. Recovered. Joseph F., aged nine years.

November 17, 1915: Admitted to Doctor Stevens's service, Episcopal Hospital. About four days ago became sick, attributed to eating some cabbage two days before. Pain in abdomen and then vomited; pain was continuous. This morning pain seemed localized to upper right abdomen and patient then began to show rapid and shallow respirations and to dart his tongue in and out. Has eaten very little since illness began, but has not been constipated.

On admission, heart not enlarged, no murmurs. Lungs, anteriorly resonant throughout, breath sounds normal posteriorly, dulness and absent breath sounds over right lower lobe up to angle of scapula. Diminished resonance over left lower lobe (Grocco's sign). Few moist râles above area of dulness on right. Liver not enlarged, great tenderness and much rigidity in right upper quadrant, no marked tenderness in right iliac fossa.

November 18, 1915: White blood count, 25,200.

November 27, 1915: Diagnosis of empyema made and transferred to surgical service. Temperature has been 102°–103° ever since admission, growing more irregular (99°–104°) since November 23d.

November 28, 1915: 500 c.c. of colon smelling pus removed by aspiration. Dyspnœa much relieved. Culture of pleural fluid: mixed growth.

November 30, 1915: Operation (Doctor Ashhurst)—gas. Patient supine, right chest projecting over edge of table. Resected 4 cm. of ninth rib below angle of scapula, evacuating nearly 500 c.c. of colon pus; undoubtedly *not* subphrenic, but in pleural cavity. No perforation of diaphragm felt. Drainage tube. Temperature fell at once.

December 3, 1915: Drainage freely.

December 12, 1915: Drainage much less.

December 22, 1915: Out of bed, temperature still down.

January 3, 1916: Went home. Small tube still in sinus and some discharge.

August 29, 1916: Examination in dispensary. Very slight scoliosis; sinus healed by February 1, 1916. Lung sounds normal.

CASE XX.—Empyema, recurrent (right). Recovered. Francis P., aged eleven years.

October 13, 1915: Admitted to Doctor Deaver's service, Episcopal Hospital. Two and a half weeks ago developed pneumonia; for past five days excessive cough and profuse expectoration. On admission, temperature, 101°. Pulse, 112. Respiration, 36. Respiration shallow. Right chest more prominent and motionless on breathing. Flatness over whole right chest, feeble breath sounds, tubular on deep breathing. Left chest

hyperresonant, few râles. Heart displaced to left: apex in fifth interspace, anterior axillary line. By aspiration, 5 c.c. foul, greenish pus—staphylococci, diplococci and streptococci.

October 14, 1915: Operation (Doctor Deaver)—ether. Resected 2.5 cm. of seventh rib left mid-axillary line; 200 c.c. of pus. Rubber tube.

October 24, 1915: Still profuse discharge. Temperature, 99°–103°.

October 29, 1915: White blood count, 14,640; lymphocytes, 44 per cent.

November 1, 1915: Transferred to Doctor Frazier's service. Dressings still require changing twice daily; tube still in. Temperature, 99°–101°.

November 8, 1915: Dressed once daily.

November 17, 1915: Drainage less.

November 26, 1915: Temperature up.

November 29, 1915: Temperature still up.

December 3, 1915: Second operation (Doctor Ashhurst)—ether. Old sinus excised by incision in long axis of patient's body. Excised 3 cm. of rib next above and 3 cm. more off anterior end of that formerly excised (almost closed). Burrowed between adherent lung and thoracic wall posteriorly, gush of fœtid thin pus. (Culture, streptococcus.) About 150 c.c. Rubber tube drain.

December 5, 1915: Foul discharge (B. pyocyaneus).

December 12, 1915: Improved.

December 13, 1915: Seen by Doctor Stevens, who diagnosed pus in extreme lower part of right pleural cavity. Circulation not very good.

January 14, 1916: Sinus is being exposed to sun daily.

January 24, 1916: Temperature much lower, drainage less.

February 1, 1916: Discharge very slight. Breath sounds good.

February 3, 1916: Discharged from hospital.

September 13, 1916: Robust and rosy. No deformity. Sinus healed solidly since March. Rib reformed. Thickened pleura, but lung normal. (Impaired resonance and fremitus and distant breath sounds.)

Case XXI.—Empyema, left. Encapsulated, with separate pleural effusion. Recovered. Joseph B., aged thirty-four years.

December 8, 1915: Admitted to Doctor Piersol's service, Episcopal Hospital.

Present Illness.—Three weeks ago pain in left chest and cough. On admission, temperature, 100°. Pulse, 92. Respiration, 20–24. White blood count, 22,000. Polynuclears, 89 per cent. Pain in lower left chest, slight cough. Not toxic. Not dyspnœic. Bulging of left antero-lateral chest wall; dull red, extremely tender and resistant, from anterior axillary line into axilla. Respiratory excursions limited over left lower thorax; vocal fremitus absent, anteriorly below level of nipple, in left axilla below this level and posteriorly below angle of scapula. Skodaic resonance over upper left lung. Traube's semilunar space seems obliterated. Flatness over left chest below nipple, also absent vocal resonance and breath sounds inaudible.

Right lung: rough puerile breathing throughout and dry râles. Left chest tapped posteriorly, turbid straw-colored fluid, not true pus. (Laboratory report: gelatinous, 320 cells per cubic mm.; 80 per cent. polynuclears; 20 per cent. lymphocytes.) Blood Wassermann negative.

December 9, 1915: Seen in consultation by Doctor Ashhurst. Evidently an extrathoracic abscess over left antero-lateral chest, and inflammatory exudate in pleura. Transferred to surgical service.

Operation (Doctor Ashhurst).—Novocain, ¼ per cent. Resected two inches of left eleventh rib posteriorly—pleura opened—profuse discharge of slightly turbid fluid. No odor. (Laboratory report: negative.) Diaphragm comes up against wound in expiration, and fluid only discharges when diaphragm is pushed away. Finger introduced finds no adhesions except where diaphragm comes against ribs. Lung not felt. About 300–350 c.c. of turbid fluid evacuated and more drains while dressing wound.

Tube inserted up four inches and stream of turbid serum escaped through it. Tube sutured to skin, and ends of skin wound closed. Culture of serum.

Patient on back. Novocain. Incision over subcutaneous (left) phlegmon—thick, yellow, inodorous pus (culture, staphylococcus). This pus has spread over area 8 cm. in diameter superficial to ribs and discharged between seventh and eighth ribs through orifice admitting little finger, into walled-off cavity about 8 cm. in diameter—encapsulated empyema necessitatis. Iodoform gauze drain.

(Query: Is free pleural fluid tuberculous? Presumably it is analogous to that in pleura in subphrenic abscess, and to that in knee in osteomyelitis of tibia.)

December 10, 1915: Temperature, 98°–103° F.

December 15, 1915: Temperature steady, 98°–99⅘°.

December 25, 1915: Tube out from posterior wound.

December 29, 1915: Out of bed.

January 7, 1916: No drainage. Went home at own request.

September 20, 1916: Examination: Returned to work two months after leaving hospital. No disability since. Both wounds solidly healed, lung normal on examination.

CASE XXII.—Empyema, right; necrosis of mandible (from noma). Died, two days (noma). Edward S., aged seven years.

December 11, 1915: Admitted to Doctor Frazier's service, Episcopal Hospital. Pneumonia at home for three weeks. December 7th refused to eat because of sore mouth, and for four days before admission was pulling out loose molar teeth with his fingers; had very foul breath. Entire alveolus of lower jaw (right and left), except under the three front teeth, is necrotic, and the noma extends on to cheeks. Right lung: dull, except over extreme apex, breathing tubular and fairly loud where dull, and fremitus increased. Left lung: numerous râles and tubular breathing at base.

1 P.M.—Red blood count, 2,800,000. Hæmoglobin, 35 per cent. White blood count, 15,800. Temperature, 101⅘°. Condition very poor. Aspiration right chest—10 c.c. thick, yellowish pus.

2 P.M.—Child desperately ill from sepsis.

5 P.M.—Operation (Doctor Ashhurst)—gas. Resection of 3 cm. of eighth right rib in posterior axillary line, patient on back. Pleura not injured. On opening it (rather thick) a squirt of thick creamy yellow pus, no odor. (Culture, mixed growth.) About 300 c.c. evacuated. Cavity extends three ribs lower, to costophrenic space, and up as far as finger can reach toward apex. Respirations very bad. (Inhalation of ammonia.) Rubber tube drain. Then fuming nitric acid to mouth, and alveolus of mandible excised.

December 13, 1915: Died, 1 A.M.

CASE XXIII.—Empyema, left. Recovered. James C., aged three years.

January 4, 1916: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Three weeks ago became ill with cough, high temperature and pain in left side. On admission, respiration rapid, expansion absent on left. Fremitus is diminished and breath sounds almost absent; entire left chest is dull on percussion. Apex beat in mid-line. White blood count, 16,640. Polynuclears, 71 per cent. 500 c.c. pus drawn by aspirator. No dyspnœa before or after. Report from Laboratory, January 5th: Pneumococcus.

January 7, 1916: Operation (Doctor Ashhurst)—ether. Patient prone, resected tenth left rib in posterior axillary line; 250 c.c. of creamy pus evacuated, no masses of lymph. Loosened lung from diaphragm and from spinal gutter, whereupon it expanded 50 per cent.

January 17, 1916: Drainage much less.

January 22, 1916: Temperature up, slight cough.

January 26, 1916: Developed measles and sent to Philadelphia General Hospital.

February 3, 1916: Returned from Philadelphia Hospital. Temperature, 100°–101°.

February 6, 1916: Temperature, 104°. Draining freely.

February 15, 1916: Temperature falling.

February 26, 1916: Tube removed.

March 1, 1916: Tube replaced.

March 6, 1916: Tube removed. No drainage.

March 10, 1916: Discharged. Sinus healed.

September 6, 1916: No scoliosis, rib reformed, perfect result.

CASE XXIV.—Empyema, encysted left. Recovered. Philip G., aged fourteen years.

December 27, 1915: Admitted to Doctor Piersol's service at the Episcopal Hospital. Two weeks ago developed chill, headache, vomited several times and had generalized slight pain. After few days felt better and went out. Then right ear began to ache; and two days ago developed cough, and became constipated. Has now coryza, cough, slight dyspnœa and is constipated. No appetite since illness began.

On admission, fairly well built, slender, not emaciated, no adenopathy. Heart is not enlarged nor displaced, apex beat in usual location, no murmurs, sounds of fair quality. Lungs are negative, good breath sounds, good resonance throughout. Abdomen negative. White blood count, 11,040.

December 30, 1915: Marked diminution in breath sounds over left base posteriorly, few sonorous râles in lower left axilla. Sputum negative for tubercle bacilli.

January 2, 1916: Widal negative.

January 4, 1916: Marked bronchial breathing over right apex posteriorly.

January 6, 1916: Blood culture—short chain streptococcus. Malaria negative White blood count, 19,960.

January 12, 1916: Dulness on percussion and great tenderness at left base posteriorly. Nothing obtained on exploratory puncture.

January 13, 1916: Examined by Doctor Ashhurst in consultation. Has been in medical ward over two weeks with septic temperature (100°–104°) and chills. Patient is extremely emaciated and anæmic. Left lung dull at base, all signs of empyema, but needle found no pus. Very tender in left costovertebral angle. Excursion of costal margin from midline (Hoover's test) not increased, no more tender in left than in right hypochondrium. X-ray (only properly interpreted after operation) shows dense shadow above left diaphragm, upper level horizontal (not domed) and higher than diaphragm on right. A diagnosis was made of encysted empyema and exploratory operation advised.

January 14, 1916: Operation (Doctor Ashhurst)—stovain, locally, for resection of rib; gas for intrathoracic explorations. Patient prone. Excised 5 cm. of tenth rib near angle. Then incised pleura. Lung did not collapse. Diaphragm exposed, soft; spleen easily palpated through diaphragm, no sign of subphrenic abscess, and on retracting upper ribs (now general anæsthetic) lung was seen to be plastered on to diaphragm. Extended incision up parallel to spine, excised 6 cm. more of rib at vertebral end. Dissected carefully with finger, raising lung from diaphragm: a flood of pus (500 c.c.) (culture, streptococci) from between lung and diaphragm (Fig. 14). Lung now collapsed. Drain, rubber tube and iodoform gauze between lung and diaphragm. Ends of incision (12.5 cm.) closed with silkworm-gut.

January 15, 1916: Given enteroclysis and liquids by mouth. Looks better, quiet, comfortable, but very weak and thin.

January 17, 1916: Temperature, 98°–100° ever since operation. Eating better. Looks better.

January 22, 1916: Tube shortened.

January 25, 1916: Tube removed. Rapidly improving.

January 31, 1916: Up in chair.

February 14, 1916: Able to walk.

February 26, 1916: Sent home.

August 9, 1916: In dispensary in good general health, sinus in wound just admits probe; has been dressed three times weekly by family physician since leaving hospital;

tube was retained until last week, since when gauze has been employed. It was recommended that the packing be discontinued.

February 29, 1920: Sinus closed permanently a week after the gauze packing was discontinued. The only symptom he complains of now is a pricking in the scar at times, on exertion. He works as meatchopper, lifting heavy portions of the carcasses without disability. There is no scoliosis and no deformity, except the very much depressed scar. The lungs are normal, but the ribs below the scar do not move in forced respiration as do those on the other side.

Case XXV.—Empyema, encysted, right. Recovered. William B., aged thirty years.

December 22, 1915: Admitted to the Episcopal Hospital, Doctor Piersol's service. Six weeks ago developed pain in back and slight dyspnœa; began to cough with slight muco-purulent expectoration. Yesterday pain in right side at costal margin, cough, some dyspnœa, rapidly becoming worse. On admission, temperature, 103°. Pulse, 104–108. Respiration, 32. White blood count, 10,000. Polynuclears, 91 per cent. Well developed, dyspnœic, alæ of nose moving in each respiration. Lungs: impairment of resonance over right side, most marked in middle and lower lobes; tactile and vocal fremitus decreased, and bronchial breathing with moist râles over middle and lower lobes. Few sonorous and crepitant râles over right upper lobe. Left lung negative. Heart normal. Liver enlarged 5 cm. below costal margin.

December 26, 1915: Temperature fell by lysis to 100°.

December 30, 1915: General condition improved. Temperature, 100°–103°. Marked bronchial breathing over middle of right lung posteriorly.

January 3 to 14, 1916: Temperature, 99°–101°–102°, irregular.

January 13, 1916: Seen by Doctor Ashhurst in consultation. In medical ward (Piersol) some weeks with sepsis following pneumonia ("unresolved").

Examination.—Right chest dull (flat) from angle of scapula down, fremitus normal, breath sounds distant. A diagnosis of empyema was made from history and condition of "unresolved pneumonia." Medical attendants could not locate pus. Needle in eighth interspace drew only blood.

January 14, 1916: Transferred to surgical ward. Dulness over middle and lower lobes right. Right base, however, transmits vocal fremitus, although voice and breath sounds are diminished. X-ray shows haziness in upper and middle lobes.

Operation (Doctor Ashhurst).—Novocain, ¼ per cent. Patient prone. Incision 10 cm. over ninth rib, excised 8 cm. Diaphragm and lightly adherent lung presented on opening pleura. Separated lung from diaphragm by finger far as could reach, no pus. Surface of diaphragm rather sensitive, lung insensitive. Then burrowed between middle and lower lobes, no pus; entirely insensitive. Then burrowed up along parietal pleura, and found very dense adhesions up along fifth to sixth ribs, finally broke through, when just about to abandon operation, and got a flood (perhaps 250 c.c.) of blood-stained pus. (Culture, pneumococcus.) Evidently had been interlobar (upper and middle lobes) and had worked out to parietal pleura (Fig. 15). Parietal pleura rather sensitive. Slight coughing on evacuation of pus, and partial collapse of lung. Drain: rubber tube up along parietal pleura for four inches.

January 15, 1916: Looks convalescent.

January 16, 1916: Drains freely.

January 20, 1916: Tube removed.

January 23, 1916: Temperature normal. Out of bed. Sputum negative for tubercle bacilli.

January 31, 1916: Convalescent.

February 3, 1916: No discharge. Small granulating wound. Sent home.

August 9, 1916: Cannot be traced.

Case XXVI.—Interlobar empyema, left (against pericardium). Died. Katherine D., aged thirty-eight years.

August 3, 1916: Admitted to Doctor Ashhurst's service, Episcopal Hospital.

Past History—Four and a half years ago had attack of pneumonia and pleurisy (left sided) and was in another hospital five months; had septic temperature and chills. Was aspirated, but no pus could be obtained.

Present Illness.—Has had no trouble since, until six weeks ago, when she had pain in left side for two days, severe pain for three-quarters of an hour, followed by chill. July 28th had temperature of 103°, pulse, 120, and sent for Doctor Shannon. Has had chills. August 1 went to bed complaining of weakness and fever and pain on left side. Last night (August 2d and 3d) had pain in right shoulder, relieved by hot-water compresses. Has had slight cough, but not much expectoration. Coughed more before taking to her bed.

On admittance, well nourished, no evidence of acute suffering. Breath foul. Temperature, 101°–105°. Pulse, 120–150. Respiration, 32–40. White blood count, 21,000. Chest: posteriorly inspection and palpation negative, except for slight restriction of expansion and decreased fremitus at base of left lung. Percussion note is impaired from seventh interspace in mid-axillary line to vertebral column and extending to base of left lung. Auscultation shows decreased breath sounds over this area. Right lung negative, breath sounds normal. Heart rapid (no murmurs), outline normal. Abdomen negative. Puncture in seventh interspace, anterior axillary line (record syringe) gave no fluid. After admittance had chill.

August 4, 1916: Another chill to-day. Temperature, 101°–104°. X-ray apparatus out of order and no röntgenological examination could be made.

August 5, 1916: Operation (Doctor Ashhurst)—novocain, ¼ per cent. Patient prone. Incision 10 cm. over left eighth rib, excised 8 cm. Opened pleura, lung lightly adherent throughout, but freely movable. Lung densely adherent at periphery to diaphragm. Packed off above. Dissected lung from diaphragm. No pus. Felt inner border of spleen through diaphragm no subphrenic abscess. Packed over diaphragm, removed upper packing and separated lung from parietal pleura. Then extended skin incision, excised 3 cm. more of eighth rib and 10 cm. of seventh; dissected with two forceps in interlobar space for 5 or 6 cm., but found only dark blood. (Culture, sterile.) Iodoform gauze stuffed in this hole and wound closed in layers (Fig. 16). (Hoped pus would break into wound as in Case XVIII.)

August 7, 1916: Examination by Doctor Hooker. On left side down to angle of scapula there are normal breath sounds, resonance and fremitus. Unable to examine below on account of dressings. Heart: second aortic accentuated, no murmurs. Blood culture negative. One chill to-day. Temperature, 98.4°–104°.

August 9, 1916: Examination by Doctor Hooker. Heart: second aortic accentuated, roughening at mitral valve, presystolic. Entire right lung shows increased vocal resonance, many crepitant râles, impaired note on percussion, typical pneumonia (hypostatic in origin). Left lung same as last examination. Drainage removed and wound cleaned.

August 10, 1916: Expression is anxious, apathetic, listless, but rather restless. Heart sounds distant. Some evidence of pus on dressings, not present yesterday. Neck held very rigid, moves extremities when attempts are made to flex neck. White blood count, 19,000; polynuclears, 79 per cent.; hæmoglobin, 55 per cent. Temperature does not fall below 101° any more. X-ray shows shadow of abscess to left of midline of thorax, under sixth and seventh ribs (Fig. 18).

August 13, 1916: Died of sepsis. Had been moribund for last three days. Wound explored after death by Dr. H. S. Spruance (ward surgeon) after removal of two more ribs. He found gauze pack had been correctly placed in interlobar space, and by separating lobes farther (about 2.5 cm.) through dense adhesions he came on abscess (100 c.c.) in interlobar space and against pericardium. Pericardium when opened was found to contain many recent fibrinous adhesions, but no pus (Fig. 17).

Case XXVII.—Empyema, left, encysted above diaphragm. Recovered. James McG., aged thirty years.

October 30, 1916: Admitted to the Episcopal Hospital, Doctor Robertson's service.

Present illness began October 19th with coryza. October 28th had chill and began expectorating; continuous pain in left chest. On admittance, signs of pneumonia at base of left lung.

November 5, 1916: Temperature falling by lysis.

November 11, 1916: Temperature has not reached normal. Puncture of chest negative.

November 12, 1916: Puncture again negative.

November 16, 1916: Third puncture reaches pus in eighth interspace, 40 c.c. of pus withdrawn. Smear and culture of fluid is negative.

November 17, 1916: Transferred to surgical ward (Doctor Ashhurst). Temperature, 99°–103° for the last week.

Operation (Doctor Ashhurst).—Eucain, 1 per cent. Excised 5 cm. of tenth rib, lung does not collapse, lightly adherent to costal pleura and densely to diaphragm. Separated costal adhesions, no pus within reach of opening. Separated lung from diaphragm as far as possible, no pus, but adhesions at limit of fingers were more dense. Excised 5 cm. of ninth rib, and after gauze pack above, dug lung away from diaphragm to dome, when about 60 c.c. of pus discharged. Drain: rubber tube wrapped in iodoform gauze.

November 18, 1916: Temperature still up.

November 22, 1916: Still draining freely. Temperature, 100°–103°.

December 1, 1916: Tube removed. Out of bed.

December 8, 1916: For continuing temperature and history of syphilis is given neosalvarsan.

December 12, 1916: Temperature normal.

December 18, 1916: No drainage; incision closing. Sent home.

January 16, 1917: Seen by Dr. Spruance, ward surgeon; healed firmly for about one month. In perfect health.

February 29, 1920: Examination; is police officer. In perfect health. Lungs normal. On palpation ribs seem to have reformed.

Case XXVIII.—Empyema, left, encysted, upper lobe. Recovered. William B., aged twenty years.

October 18, 1916: Admitted to the Episcopal Hospital, Doctor Robertson's service. Blind since illness with pneumonia at five years of age. Onset of present illness, October 16, with chilliness, during evening; awoke during night with pain in left chest. On admission, pneumonia over upper left lobe.

October 23, 1916: Temperature falling by lysis.

November 4, 1916: Temperature has not reached normal. Signs of fluid. Puncture below angle of scapula negative.

November 16, 1916: Signs of fluid still present now confined to upper lobe, left. Puncture between angle of scapula and spine gives 30 c.c. pus. Sent to laboratory. Previous punctures all over left chest had been negative.

November 17, 1916: Seen by Doctor Ashhurst in consultation. Has been in ward over four weeks with continued temperature following typical pneumonia (left upper lobe), which declined by lysis over three weeks ago. *Repeated* punctures negative until yesterday pus was found (30 c.c. easily drawn) in fourth interspace at spine of scapula. Transferred to surgical service (Doctor Ashhurst).

Operation.—Eucain, 1 per cent. (Patient blind since five years and neurotic.) Excised 4 cm. of fourth and fifth left ribs between scapula and spine, flood of curdy pus and great handfuls of coagulated lymph. Cavity extends to first rib and down only to sixth or seventh in mid-axillary line, evidently encapsulated over interlobar fissure. Drain: large rubber tube and iodoform gauze (Fig. 19).

November 18, 1916: Temperature falling, profuse drainage.

December 27, 1916: Up and about ward part of day.

December 30, 1916: 50,000,000 autogenous vaccins, given with idea of hastening closure of cavity.

January 8, 1917: 100,000,000 vaccins given.

January 16, 1917: Still in ward, not much discharge. Temperature normal for one week, until to-day, sudden rise.

January 26, 1917: Temperature about normal. Scarcely any discharge. Up all day.

January 27, 1917: Sent home. Scarcely any discharge.

February 21, 1920. Three years after leaving hospital, examination shows him fat and healthy (Fig. 20). The sinus did not heal entirely for nearly ten weeks after his discharge (over four months after the operation). Examination shows the lungs normal. He has full use of his left shoulder, in spite of section of trapezius and rhomboid muscles at time of operation. As far as can be felt the ribs have reformed. His only complaint is of a pricking feeling in the scar when he puts his hand to small of back.

CASE XXIX.—Empyema, left (two distinct encysted empyemas). Recovered. Johan L., aged twenty-three years.

December 28, 1916: Admitted to the surgical ward from medical ward with post-pneumonic empyema. Temperature, 102°. Originally admitted to Doctor Deaver's service, October 6, 1916, with diagnosis of syphilis, having contracted mixed chancre about one month previously, followed in two weeks by suppurative left inguinal adenitis, there being unhealed sinuses here on admission. November 6th, transferred to medical ward (Dr. A. A. Stevens) for constitutional treatment for syphilis, local lesions being under control.

December 4, 1916: Developed pneumonia at base of left lung.

December 15, 1916: Temperature falling by lysis.

December 18, 1916: Temperature has not reached normal. Signs of fluid; 100 c.c. of cloudy fluid at second puncture, at angle of scapula. (Culture, pneumococcus.)

December 21, 1916: Flatness extends almost to spine of scapula; heart displaced 2.5 cm. toward right.

December 23, 1916: On puncture a small amount of pus recovered from depth at left scapular angle. (Culture, diplococci.)

December 25, 1916: No improvement.

December 27, 1916: Temperature slightly higher (101°–103° since December 24th).

December 28, 1916: Transferred to surgical ward (Doctor Ashhurst) with diagnosis of empyema.

December 29, 1916: In medical ward many weeks with pneumonia, after this temperature reached normal, then rose again and for a couple of weeks has been high and irregular. Five recent punctures negative where signs indicated fluid. Yesterday puncture in eighth interspace below angle of scapula drew 40 c.c. pus (pneumococcus).

Operation.—Eucain, 1 per cent. Patient prone. Resected 5 cm. of eighth or ninth left rib and pus oozed before it was removed; pleura nevertheless thick (2 mm.); pus thick, creamy, many curds. Cavity about 8 to 10 cm. in diameter on diaphragm and against costal pleura. Burrowing in adhesions opened *another pocket of pus* above and against ribs. Cavity extended upward toward interlobar region. Lower lobe densely adherent to diaphragm at one point, loose all around. This piece of lung was dissected from diaphragm. Considerable complaint and coughing when costal pleura was rubbed in burrowings, but lung and diaphragm insentitive. Drain: rubber tube and iodoform gauze. No ligatures. No sutures.

January 15, 1917: Drainage lessening.

February 2, 1917: No discharge. Temperature normal. Wound granulating. Up and about ward.

February 9, 1917: Discharged.

February, 1920: The Swedish Consul at Philadelphia reports that this patient was last seen by him about one year ago, apparently in good health.

CASE XXX.—Empyema, left. Died after five weeks (sepsis from thoracic wound). Robert S., aged seven years.

January 17, 1917: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Had had pains in left side and stomach for a week or ten days; January 5th took his bed; vomited; had cough, chills and high fever. Treated at home for twelve days for bronchopneumonia. Family physician diagnosed empyema following pneumonia.

January 17, 1917: On admission, an anæmic, poorly nourished boy. Has incomplete cleft of palate; had operation for harelip as infant. Expansion of left chest limited. Palpation negative, dulness and distant breath sounds. *Puncture negative.*

January 18, 1917: Operation (Doctor Ashhurst)—eucain, 1 per cent. Patient prone. Resected 4 cm. left eleventh rib, posteriorly. Opened pleura, flood of reddish brown pus (culture, diplococci). Tube passed upward four inches and held in by silkworm-gut suture. Lung could not be felt. Time, fifteen minutes.

January 23, 1917: Temperature normal until to-day. Tube removed. Out of bed.

January 25, 1917: Draining slightly. Temperature normal to 100° since removal of tube.

January 26, 1917: Up in chair all day, no discharge.

January 27, 1917: Wound healing well.

January 31, 1917: Temperature rose to 104° suddenly. Transferred to Doctor Mutschler's service.

February 2, 1917: Vomits, no drainage. Temperature, 100°–103°.

February 3, 1917: Wound explored with hæmostat and several ounces of pus obtained. Tube replaced. Temperature fell to normal.

February 5, 1917: Draining well.

February 7, 1917: In good general condition. Temperature normal. Slight drainage of purulent fluid from tube. Cavity irrigated with normal saline. This causes some pain. Fluid comes back clear. Temperature rose to 100⅗°.

February 9, 1917: Appetite excellent, only slight discharge, but child is fretful and nervous. Daily irrigations.

February 12, 1917: Temperature, 98°–100°.

February 15, 1917: Temperature rose suddenly to 103° and continued hectic 100°–104° until death, February 23. Daily irrigations.

February 18, 1917: Septic temperature with sweats. No chills. Vomited. Unproductive cough. Small area of cellulitis around drainage tract with extreme tenderness. Chest negative, but no pus draining. Pulse rate now 120–140, formerly 80. Respiration, 28–32, formerly 20. Slightly impaired percussion note over left chest posteriorly, two punctures negative. Irrigations continued.

February 20, 1917: No drainage. Unable to sit up without support, will not eat. Vomits at intervals. A maculo-papular rash like measles over trunk and extremities. Throat negative. Irrigations continued.

February 21, 1917: Sweats, paroxysms of vomiting without other abdominal signs or symptoms, anorexia and increasing weakness continue.

February 22, 1917: Irrigated. Tract is open, but there is no drainage. Marked abdominal distention but no pain or rigidity. Rash still present. Temperature falling.

February 23, 1917: Died with acute dilatation of heart, 11:30 A.M. Temperature subnormal. Cause of death: Sepsis from wound of thoracic wall.

CASE XXXI.—Empyema, right. Died (sepsis from thoracic wound). Stanley K., aged nine years.

January 15, 1917: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Present illness began December 24, 1916, with chill, fever, vomiting and jaundice. Four days later, pain in right side radiating to shoulder and to left chest, finally localized to right costal margin. Short of breath and breathing pained him. Was

treated for lobar pneumonia. Two days before admission mother noticed right chest was swollen, called doctor's attention to it, and he found pus on puncture.

On admission, right lower chest had interspaces obliterated, flat, absent breath sounds, apparently massive empyema, two punctures in axilla, negative, but pus found below angle of scapula (Doctor Spruance, ward surgeon); 200 c.c. pus removed by puncture. Smear and culture negative. Polynuclears, 55 per cent.; lymphocytes, 45 per cent.; temperature, 101⅘° F.; pulse, 160; respiration, 42.

January 16, 1917: Operation (Doctor Ashhurst)—eucain, 1 per cent. Patient prone. Resected 4 cm. of eleventh right rib. Pleura opened just above its reflection on to diaphragm. No pus found here. Inserted finger and in adhesions above tenth rib, close to spine, got a gush of pus (culture, negative). Pus reddish brown, no curds. Cavity runs up spinal gutter as far and as wide as finger can reach. Inserted rubber tube upward 10 cm. and more pus then spurted when he coughed. (One litre in all.)

January 21, 1917: Draining considerably.

January 22, 1917: Tube removed.

January 25, 1917: Still draining. Out of bed on house diet. Temperature, 99°–100°.

January 26 to 31, 1917: Temperature, 99°–103°. Pus probably dammed up.

January 31, 1917: Transferred to Doctor Mutschler's service.

February 1, 1917: Wound closed. Temperature irregular.

February 1 to 5, 1917: Temperature, 99°–103° F.

February 7, 1917: Operation wound granulating, no discharge. On deep inspiration (which brings on coughing) there is limitation of expansion on right side above. Tactile fremitus absent and breath sounds distant in right scapular region. At right base many moist bubbling râles. Percussion note hyperresonant except in scapular region where it is flat.

February 6 to 8, 1917: Temperature, 99°–102°.

February 8, 1917: Finger inserted in old sinus (no anæsthetic) base of lung felt to crepitate and expand with inspiration. Adhesions bound an area from the ninth rib above, in front by posterior axillary line, below by diaphragm. These adhesions cannot be broken by gentle pressure and more is not attempted because of fear of pleural reflex. Temperature fell from 102° to 99°.

February 10, 1917: Dyspnœa increasing. Drenching sweats twice daily. Well-defined area of flatness extending from scapular spine to its angle and as far forward as mid-axilla. Puncture in sixth interspace at angle of scapula, and creamy purulent fluid is withdrawn about the consistency of molasses in March. A second futile attempt is made to drain this encysted collection of pus through the old incision.

February 9 to 12, 1917: Temperature, 99°–101° F.

February 12, 1917: General condition excellent.

February 13, 1917: Operation (Doctor Mutschler)—ether; 3 cm. of seventh right rib resected in mid-scapular line, and 2 oz. (60 c.c.) of creamy, purulent, viscid fluid evacuated. Pleural cavity is then irrigated with sterile water until latter comes away clear. Rubber tube inserted 6 cm., stitched to skin by silkworm gut. Incision is then closed with interrupted silkworm-gut sutures and dressing applied. On return to ward able to breathe comfortably in recumbent position.

February 14, 1917: Dressings soaked with purulent blood-stained discharge. Old tract has definitely closed. Pleural cavity is irrigated with warm normal saline solution, injected through rubber drain tube and again withdrawn.

February 15, 1917: Dressings and irrigation repeated. Unable to sit up without excruciating pain in right scapular region. The wound is draining well, but temperature continues elevated (100°–103°).

February 17, 1917: Drainage less. Well-defined area of induration, redness, tenderness, swelling and œdema around drainage tube. Irrigation is followed by a fit of coughing and fluid expelled is seen to contain small pieces of coagulated exudate.

February 17 to 20, 1917: Temperature, 100°–104°, and so on until death, February 25.

February 19, 1917: Pulse feeble, rapid but regular. Vomited. Takes liquids sparingly, no food. Macular rash, coppery in color, over limbs and trunk. Is evidently exceedingly toxic, wound drains little. Rubber tube removed, irrigation performed. Percussion note is resonant to level of ninth rib, below this there is flatness, with distant breath sounds. Puncture in ninth interspace causes no pain, clear, colorless fluid, evidently residue of irrigating fluid, is withdrawn; contains small fragments of floating exudate.

February 20. Obviously very ill. No appetite, listless all day long. Area of cellulitis around tube is larger. Movements are painful, as if they involve the muscles in this area. Sutures removed.

February 24, 1917: Vomited large quantity of greenish material. Dyspnœic, toxic, dull and drowsy. Toward evening cyanotic.

February 25, 1917: Died, 3:30 A.M. Cause of death: Sepsis from wound in thoracic wall.

CASE XXXII.—Empyema, right. Recovered. Whitman L., aged forty-six years.

January 3, 1917: Admitted to the Episcopal Hospital, Doctor Stevens's service. Has had a cough for years. Last evening was taken with chills and sweats, vomiting and severe headache. This morning had general pains and pain on inspiration in right chest. Typical pneumonia at right base.

January 12, 1917: Signs of fluid at right base. Temperature has never reached normal (99°–101°).

January 14, 1917: Developed bilateral suppurative otitis media. Paracentesis of right chest negative.

January 17, 1917: Temperature rising (101°–103°). Still signs of fluid at right base. Is not septic.

January 22, 1917: Signs of fluid at right base, puncture gave pus (culture and smear, diplo-bacillus).

January 23, 1917: Puncture draws 250 c.c. creamy pus.

January 24, 1917: Transferred to surgical service (Doctor Ashhurst). Operation (Doctor Ashhurst)—eucain, 1 per cent. Patient prone. Resected 4 cm. of eleventh rib, found very much thickened pleura, cut through it and exposed fibres of diaphragm. Then cut higher in pleura (costo-phrenic sinus) and opened pleural cavity whence over 500 c.c. of pus was discharged, creamy, inodorous. (Culture, diplococcus and pneumococcus?) Diaphragm not sensitive, costal pleura very sensitive. Cavity extends up and back beyond reach of finger. Drain: large rubber tube up for 10–12 cm.

January 26, 1917: Drainage almost stopped. Sat up in chair awhile.

January 31, 1917: Still draining profusely. Temperature normal ever since operation.

February 10, 1917: Less drainage. Learning to walk.

February 12, 1917: Tube removed.

February 17, 1917: Went home.

February 29, 1920: Wound healed firmly four weeks after leaving hospital. No disability. Lungs normal.

CASE XXXIII.—Empyema, right. Recovered. John B.; aged twenty-six years.

January 10, 1917: Admitted to the Episcopal Hospital, Doctor Stevens's service. Illness began January 4th with chill and sweat, and pain in right side; has had sweat almost every night. On admission exceedingly toxic and sick, entire body deeply jaundiced. Cyanosis of finger tips. Pneumonia at base of right lung.

January 12, 1917: Has been markedly delirious. Strapped to bed. Temperature low, never up to 101°, evidently poor reaction.

January 16, 1917: Rational. Jaundice subsiding.

January 23, 1917: Signs of fluid at right base (apical pneumonia). Puncture gives pus. Temperature, 101°–103° since January 21st.

January 24, 1917: Transferred to surgical ward (Doctor Ashhurst). Operation—eucain, 1 per cent. Patient prone. Resected 4 cm. of eleventh rib, pleura thickened, pus gushed on opening it. Finger introduced found opening at lowest limit of cavity, which extended up and back beyond reach of finger, limited in front and below by the attachments of diaphragm. About 1 litre of curdy yellow pus evacuated (culture, pneumococcus) and drains profusely when tube is inserted, up 10–12 cm. along spine.

January 26, 1917: Drainage almost stopped. Sat up in chair awhile.

January 31, 1917: Still draining profusely.

February 6, 1917: Sits up each day. Temperature, 98°–102°.

February 12, 1917: Tube removed, temperature having reached normal.

February 18, 1917: Discharged. Temperature normal since last note. Patient not traced.

CASE XXXIV.—Empyema, right. Recovered. William D., aged sixteen years.

January 9, 1917: Admitted to the Episcopal Hospital, Doctor Stevens's service. Chief complaint: pain in right chest worse on coughing and on deep inspiration. Temperature, 103°. Onset January 5th with chill and sweat; cough, blood-stained sputa; headache and weakness. Excessively ill with typical lobar pneumonia, right middle lobe.

January 11, 1917: Type II pneumococcus reported in sputum.

January 12, 1917: Pericardial friction sounds. Very toxic. Abdomen markedly distended. Temperature fell by crisis to 99°.

January 16, 1917: Temperature has gradually risen again to 103° F. Pneumonia at *left* base.

January 21, 1917: Still very ill. Temperature irregular, 100°–103°. Sweats at night. Signs suggestive of fluid at right base. Consolidation at left base.

January 25, 1917: 250 c.c. pus drawn by puncture in ninth interspace, posterior right side.

January 26, 1917: Transferred to surgical service (Doctor Ashhurst). Temperature fell to normal after puncture. Is blue, thin, anæmic. Right chest (lower) is flat, distant breath sounds, somewhat diminished fremitus.

Operation (Doctor Ashhurst).—Eucain, 1 per cent. (10 c.c. only). Patient prone. Resected 4 cm. of eleventh right rib, pus oozed before rib was resected. About 500 c.c. creamy yellow pus evacuated. (Culture, diplococcus, pneumococcus.) Finger introduced felt no lung or adhesions. Opening was at bottom of coto-phrenic sinus. Large rubber tube passed up 8–10 cm. and held to upper skin margin by silkworm-gut suture. Time, ten minutes.

January 27, 1917: Temperature normal.

January 31, 1917: Temperature irregular, 98°–102°.

February 3, 1917: Still draining freely.

February 6, 1917: Out of bed daily. Temperature still irregular.

February 12, 1917: Smaller tube inserted.

February 18, 1917: Temperature normal. Free drainage. Learning to walk.

February 19, 1917: Tube removed.

February 21, 1917: Wound closes between daily dressings, damming up a few drops of pus.

February 22, 1917: Small tube reinserted. Went home.

February 21, 1920: Three years after operation. Scar has remained healed since discharge from hospital. Then weight was eighty pounds. In four weeks it reached one hundred and fourteen pounds, and is now steady at one hundred and twenty pounds. Went to State Sanitorium for Tuberculosis on discharge and remained there for nearly a year. Lungs are normal on examination, except slightly distant breath sounds over right chest posteriorly. Chest expands normally. Well-developed chest. No deformity. Rib has reformed. Some evidence of old endocarditis audible, and gets out of breath easily, but able to do a man's work.

CASE XXXV.—Empyema, right (two distinct encysted empyemas). Recovered. Edward H., aged thirty-two years.

April 7, 1917: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Referred by Dr. H. R. M. Landis. Taken ill ten weeks ago. Diagnosis: pneumonia, right. Sent to a hospital and after about seven weeks as he did not get well they said he was tuberculous (called it "unresolved pneumonia") and sent him to White Haven, Pa. Seen there March 17, 1917 (three weeks ago), by Doctor Landis, who drew off a quantity of *sterile* pus, and referred him to Doctor Ashhurst.

Examination.—Emaciated, free expectoration of muco-pus (not fœtid). Right chest dull over lower lobe and signs of fluid. Needle drew from eighth interspace in posterior axillary line, 350 c.c. of pus (92 per cent. polynuclears; smear, negative).

April 8, 1917: Operation (Doctor Ashhurst)—eucain, 1 per cent. Resected 5 cm. of ninth right rib. Patient prone. The pleura much thickened (2–3 cm.). Spurting intercostal artery tied by suture, caused much pain, checked by eucain injection. On opening pleura pus escaped, from *thoroughly walled off abscess between diaphragm and lung.* Diaphragm felt normal, soft, depressible; and smooth liver palpable beneath it. The upper margin of cavity was formed by lung, densely adherent at periphery to costal pleura, just above ninth rib. Diaphragm rose to tenth rib. Suprаphrenic abscess extended four inches toward dome of diaphragm. With much difficulty but *without causing any pain,* adhesions of lung to costal pleura were broken through by the fingers, first far posteriorly, and then lung was peeled off the costal pleura all the way front (here it was painful, under anterior costal margin, eighth, seventh and sixth ribs) (Fig. 21).

It was noted that the needle puncture which drew pus in the eighth interspace was above the level where the lung was adherent to the chest wall. *On detaching the lung a perfect flood of pus (over 500 c.c.) came from above, from between the lung and ribs;* this cavity extended up as far as finger could reach. (Culture, diplococcus.)

Drain: rubber tube between lung and diaphragm and another (2 cm. lumen) between lung and costal pleura, running up toward apex 15 cm. Iodoform gauze stuffed in wound between these two tubes to hold margin of consolidated lung away from wound and convert the two abscess cavities into one. Wound not sutured (12.5 cm. incision). Time, thirty minutes.

May 27, 1917: Uneventful convalescence. Wound has been merely moist for last two weeks.

June 3, 1917: Went home. Wound now closed for a week with normal temperature, though up to then small tube entered six inches easily. Weight, one hundred and fifty pounds, gain of twenty-three pounds in eight weeks since operation. Breath sounds normal. No symptoms. Right chest moderately contracted in front.

July 6, 1917: Reports he will return to work in one week.

November, 1917: Sinus discharged but healed in three weeks.

February, 1919: Empyema formed again and another operation, 5 cm. above former operation, done by Doctor Schell at Northwestern General Hospital. Recovered. Weight, one hundred and seventy pounds.

September, 1919: Moved to Denver. Since then one reaccumulation was opened; healed in six weeks.

March, 1920: Weight, one hundred and seventy pounds. Excellent health, no symptoms of tuberculosis, working every day.

CASE XXXVI.—Empyema, right, encapsulated. Recovered. Oliver C., aged twenty-one years.

July 9, 1919: Admitted to the Episcopal Hospital, Doctor Robertson's service. Ill since July 7th, typical pneumonia. Chief complaint is pain in right chest and shortness of breath. On awakening in morning had chill, became feverish and dyspnœic, and developed pain in right chest on inspiration. On admission: signs of pleuro-

pneumonia at right base. Temperature 100°–102°. Thought to have fluid, but puncture negative.

July 12, 1919: Puncture in posterior axillary line is negative.

July 14, 1919: Consolidation also of left base.

July 15, 1919: Pleuro-pericardial friction sounds on left.

July 24, 1919: Temperature continues 99°–103°.

July 25, 1919: Temperature declined by lysis, yesterday shot up again to 103°. Needle in ninth interspace in scapular line drew 350 c.c. pus. (Culture, pneumococci.) Transferred to surgical service (Doctor Ashhurst).

July 26, 1919: Right empyema encapsulated between lung and diaphragm. Operation (Doctor Ashhurst)—novocain, ¼ per cent. Excision of 5 cm. of tenth rib. Costal pleura normal but adhesions of lung to costal pleura above and in front of opening, and lung adherent to diaphragm. Could not be separated by fingers. Packed. Lung cut loose from diaphragm by scissors; 150 c.c. yellow, creamy pus. Tube and gauze wick. A few whiffs of ether toward end. Diaphragm felt normal for 5 cm. from ribs, then felt hard; this part was adherent lung.

August 3, 1919: Temperature has reached normal. Little discharge.

August 13, 1919: Tube removed. Has been up in chair several days and rapidly getting fat.

August 14, 1919: Temperature shot up to 103°. Tube replaced.

August 18, 1919: Last night and night before had chills; resident's attempts to get tube in far enough apparently ineffectual. Therefore, to-day, under gas, Doctor Ashhurst put finger into sinus, and found lung again adherent to diaphragm, and tore it loose, giving exit to bloody pus; and put tube again deep in wound, almost to dome of diaphragm.

August 25, 1919: Temperature has gradually reached normal. No discharge.

September 5, 1919: Tube removed. No discharge.

September 6, 1919: Temperature shot up to 103°. Tube replaced.

September 8, 1919: Temperature normal.

September 14, 1919: Tube removed.

September 15, 1919: Temperature shot up to 102°. Tube replaced.

September 17, 1919: Temperature normal. No discharge.

September 20, 1919: Out of bed.

September 27, 1919: Temperature stays normal, but sinus does not heal, though there is practically no discharge. Tube retained.

October 10, 1919: Tube shortened.

October 15, 1919: Tube removed and gauze wick inserted.

October 21, 1919: Bismuth paste injected to determine size of empyema cavity. X-ray shows sinus extends up to fifth rib anteriorly. (X-ray 1224 D.)

October 25, 1919: No discharge from wound since injection of bismuth.

October 30, 1919: Wound is firmly healed. Went home.

November 17, 1919: Returns on visit. Still healed. No symptoms.

February 29, 1920: Weight, one hundred and seventy pounds (one hundred and twenty-seven pounds on leaving hospital). Went to work the day after discharge from ward, and has been at work ever since. No disability. Lungs normal, except distant breath sounds at right base posteriorly. The rib has not yet reformed.

CASE XXXVII.—Empyema, right, massive. Recovered. Isador M., aged eleven years.

August 4, 1919: Admitted to the Episcopal Hospital, Doctor Robertson's service. Onset July 31st. On admission very ill, lobar pneumonia at right apex.

August 6, 1919: Puncture at angle of scapula gives 12 c.c. turbid fluid.

August 8, 1919: Puncture in axilla through fourth interspace and at angle of scapula in eighth interspace; 3 c.c. of slightly turbid pus obtained from each. Patient is better.

August 9, 1919: Puncture just lateral to angle of scapula gave 500 c.c. thick green pus. Much better after evacuation.

August 12, 1919: Puncture gives 350 c.c. of green pus. Temperature declining by lysis. Smear shows diplococci of pneumococcus type.

August 14, 1919: Temperature, 98°–102°.

August 16, 1919: Puncture gives 250 c.c. of pus, very thick and green; 20 c.c. of formalin in glycerine injected. Temperature fell from 103° to 99°.

August 18, 1919: No change. Examination by Doctor Ashhurst. Skodaic resonance over right apex anteriorly; interspaces on right side of chest obliterated. Dull in axilla and posterior to line of nipple, and posteriorly dull below the fourth interspace except close to vertebral column. Breath sounds distant. Post-pneumonic empyema. Duration, ten days. Transferred to surgical service (Doctor Ashhurst).

Operation (Doctor Ashhurst)—novocain, ¼ per cent. Resection of 4 cm. of tenth right rib posteriorly. One litre of pus (culture, pneumococci) evacuated on opening pleura. The cavity extended from diaphragm as high as finger could reach and also between diaphragm and lung (Fig. 22). Diaphragm was covered with thick gray slough, and the lower lobe of the lung stood out surrounded by pus on both phrenic and costal surfaces.

August 22, 1919: Free drainage. Doing very well.

September 1, 1919: Wound closing. Less discharge.

September 9, 1919: Tube removed.

September 13, 1919: Incision healed. Sent home.

March 21, 1920: Seven months after operation. Has gained fourteen pounds in weight since leaving hospital—present weight, eighty-three pounds. Lungs are normal, and rib appears to have reformed.

Case XXXVIII.—Empyema, right. Recovered. Evelyn S., aged nineteen years.

November 16, 1919: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Sent from Philadelphia Hospital for Contagious Diseases, where she was taken October 31st for suspected diphtheria.

October 29, 1919: Onset with sore throat and headache.

October 31, 1919: Taken to Hospital for Contagious Diseases.

November 5, 1919: Pain in right side. Dulness.

November 13, 1919: Evidences of fluid in right chest. Puncture drew 20 c.c. yellowish purulent fluid (sterile). Throat has given nine negative cultures, never a positive culture.

On admission, chief complaint is pain in both arms and back. There is subpectoral abscess below right clavicle. Patient emaciated and very septic. Temperature, 103° F.

November 17, 1919: Seen by Doctor Fussell, who advised aspiration over right base, where signs were suggestive of fluid.

November 18, 1919: Dulness and distant breath sounds over lower posterior chest. Puncture draws pus. Diagnosis: right empyema.

Operation (Doctor Ashhurst)—novocain ¼ per cent. Resection of 3 cm. of ninth rib, below angle of scapula. Pleura thickened; 250 c.c. of greenish yellow, creamy, malodorous pus evacuated (same as by aspiration). Culture, no growth. Smear, organisms like streptococci but smaller. Cavity extends backward to spine and upward beyond reach of finger. Rubber tube sutured in wound. Right subpectoral abscess to be treated later. Diaphragm came just to level of rib resection, and felt flat across top to spine, whole cavity appears rigid, and may require secondary operation for closure. Still very ill (Fig. 23).

November 19, 1919: Had a chill.

November 20, 1919: Improving, free drainage of pus.

November 22, 1919: More drainage than usual from tube. Subpectoral abscess much smaller. Still very annoying cough.

November 25, 1919: Second operation. Large abscess from beneath left scapula drained under local anæsthesia. (Smear, diplococci and staphylococci.) First sign of this abscess developed seventy-two hours previously. Still very ill.

November 30. The subpectoral abscess has refilled.

Third Operation.—100 c.c. of pus from beneath right pectoralis major drained under local anæsthesia. (Smear, diplococcus and bacillus. Culture, no growth.)

December 1, 1919: Better. Still coughing. Incision in left back almost healed.

December 20, 1919: Out of bed in wheel chair.

January 9, 1920: Tonsils removed under local anæsthesia.

January 28, 1919: All incisions now firmly healed; the last to close was that of subpectoral abscess. Empyema incision has been healed for two weeks. Went home to-day.

March 21, 1920. Four months after operation. Incision has remained closed. Is round-shouldered, with slight scoliosis convex to the left. Gets tired easily.

Case XXXIX.—Empyema, right. Recovered. Gertrude W., aged four years.

December 5, 1919: Admitted to the Episcopal Hospital, Doctor Ashhurst's service. Had whooping-cough and influenza in fall of 1919. Four weeks ago she began vomiting; physician made a diagnosis of congestion of lungs. Later he diagnosed pleurisy. On admission, anæmic, run-down child, with respiratory embarrassment. Respiratory movements almost absent on right. There is markedly diminished tactile and vocal fremitus of whole right chest, anteriorly and posteriorly. Percussion shows flatness over entire right chest. Puncture draws 75 c.c. of thick, greenish pus.

December 6, 1919: Operation (Doctor Moore, ward surgeon)—novocain, 1 per cent. Resection of 3 cm. of right ninth rib below angle of scapula 100 c.c. pus evacuated. (Smear, diplococci and staphylococci. Culture, mixed.)

December 24, 1919: Rise in temperature. Longer tube inserted.

December 27, 1919: Temperature down.

December 29, 1919: Tube removed.

January 7, 1920: Rise of temperature for past three days; 300 c.c. pus evacuated by forceps in sinus. Tube replaced.

January 13, 1919: Temperature normal since replacing tube. Tube removed to-day.

January 22, 1919: Out of bed for four days. Sent home, with incision healed.

April 11, 1920: Four months after operation; in good health; lungs normal. About four weeks after leaving hospital became feverish, and family physician picked a scab off the incision and evacuated a little pus; firmly healed again in three or four days.

Case XL.—Empyema, left. Recovered. Thomas W., aged sixteen months.

December 31, 1919: Admitted to the Episcopal Hospital, Doctor Carson's service. Eleven days ago child developed fever and cough; physician called, who diagnosed pneumonia. After convalescence fever recurred, and physician suspected empyema. On admission, there is limited expansion of left chest, no fremitus on palpation, and dulness over entire chest continuous with heart dulness. Hyperresonance over right lung. Heart displaced 2 cm. to right. Very slight change in breath, sounds are clear and distinct. Has a cough.

Puncture of left pleura gives 100 c.c. turbid straw-colored fluid, no odor. (Smear and culture show diplococci.)

January 2, 1920: Not sleeping well; dyspnœic. Pulse not so good. Puncture gives 250 c.c. turbid odorless greenish straw-colored fluid; some relief of dyspnœa.

January 4, 1920: Slightly better. Fluid collecting again.

January 7, 1920: Puncture gives 150 c.c. greenish yellow, turbid fluid, no odor. Gradually improving.

January 8, 1920: Restless and dyspnœic. Very irritable. Seen by Doctor Ashhurst in consultation. Transferred to his surgical service.

January 9, 1920: Operation (Doctor Ashhurst)—novocain, ¼ per cent. Resection of 2 cm. of tenth left rib, midscapular line. Pleura 25 mm. thick. Thin serous pus evacuated. Cavity extends as far as finger can reach. Tube drainage.

January 12, 1920: Temperature normal since yesterday.

January 14, 1920: Tube removed. No pus has drained since operation.

January 29, 1920: Temperature rose to 104° yesterday, after being nearly normal since last note; 10 c.c. of pus evacuated from sinus by inserting forceps. Then temperature fell to normal. Tube replaced.

January 31, 1920: Transferred to Doctor Neilson's service.

February 19, 1920: Temperature has been normal four days after another flare up relieved by evacuating a little pus.

February 25 to March 9, 1920: Temperature, 100°–102° F.

March 9, 1920: Not doing well. Septic temperature. Nothing definite in chest findings, but X-ray examination indicates presence of pus. Under ether anæsthesia Doctor Hawfield (chief resident physician) ran finger into sinus, but found no pus. Tube left in sinus.

March 12, 1920: Free discharge of pus.

March 20, 1920: Temperature normal for past week.

March 26, 1920: Sent home, with tube still in sinus.

April 2, 1920: Tube out. Tiny sinus.

April 9, 1920: Wound healed.

CASE XLI.—Empyema, right. Recovered. Alice M., aged twenty-nine years.

February 6, 1920: Admitted to the Episcopal Hospital, Dr. J. B. Carson's service. Chief complaint: pleurisy pains. Eight days ago was taken suddenly with chill and feverishness. Some cough. Three days ago severe pain in right chest, constant since that time. On admission, consolidation, base right lung, with skodaic resonance above.

February 8, 1920: Puncture of right chest gives 250 c.c. serous fluid. (Smear shows a streptodiplococcus, probably pneumococcus. Culture is contaminated.)

February 10, 1920: Condition fair. Temperature falling by lysis.

February 18, 1920: Temperature since February 12th has been rising gradually, and since the 14th has been 100°–103°. Transferred to surgical service of Doctor Mutschler.

February 20, 1920: Operation (Doctor Ashhurst)—novocain, ¼ per cent. Patient prone. Resection of 4 cm. of eleventh right rib. Nearly 500 c.c. of creamy, yellow pus evacuated upon opening pleura, which was 2 or 3 mm. thick. (Culture, pure pneumococci.) Finger introduced palpates no lung as high as it can reach. Opening is at a level of diaphragm, which is flat, not domed, as normally. Large tube drainage.

March 5, 1920: Tube replaced by smaller one. Very little discharge. Up and about ward. Temperature has slowly fallen to normal.

March 19, 1920: Up and about ward. Very little discharge.

March 30, 1920: Tube removed.

April 14, 1920: Went home, wound healed in several days.

CASE XLII.—Empyema, right. Recovered. Theresa D., aged twenty-seven years.

February 7, 1920: Admitted to the Episcopal Hospital, Doctor J. B. Carson's service. Chief complaint: cough, backache and pain in the chest. Husband has pneumonia and three children have influenza. Patient has been sick in bed four days. Has a nursing baby.

Examination.—Limitation of expansion and impaired resonance over *left* chest.

February 11, 1920: Improved.

February 14, 1920: Not so well.

February 15, 1920: Consolidation at *right* base.

February 18, 1920: Signs of fluid at right base. Two punctures negative; third puncture found sero-pus. (Culture, pneumococci.) Transferred to surgical service of Doctor Mutschler.

February 20, 1920: Examination by Doctor Ashhurst. Entire right chest posteriorly is flat from spine of scapula down to base, and breath sounds are distant.

Operation (Doctor Ashhurst)—novocain, ¼ per cent. Patient prone. Resection of 4 cm. of tenth right rib near its angle. Pleura slightly thickened. Upon opening

ANALYSIS OF 43 OPERATIONS FOR EMPYEMA

No.	Name, age, date of operation	Side affected	Duration of illness before operation, including a preceding pneumonia	Metapneumonic or tuberculous; and *Culture*	Massive or encapsulated	Operation and Anæsthetic	Result	Sinus healed	Remarks
1	John C., 5 years Sept. 22, 1906	R	6 weeks +	Metapneumonic, pneumococci	Massive	Eighth rib. Ether. Pus, creamy, pale yellow, sour	Recovered	Under 10 months	
2	Joseph S., 5 months July 26, 1907	L	3 weeks +	Metapneumonic, diplococci	Massive	Eighth rib. Ether. Pus, thick, inodorous	Died	.....	Death in 1 week from enteritis.
3	Ed. W., 11½ months July 25, 1908	L	2 weeks +	Metapneumonic, diplococci and staphylococci	Massive	Eighth rib. Ether, 100 c.c. thick, creamy pus	Died	.....	Death in 2 weeks from pneumonia of other lung.
4	Kath. P., 6 months July 24, 1909	L	4 weeks +	Metapneumonic, pneumococci	Massive	Intercostal incision, 7th interspace; pus 500 c.c. Ether. Rib resected 1 month later	Died	.....	Death in 2 months from sepsis.
5	Jos. T., 6 years July 24, 1909	L	5 weeks +	Metapneumonic, culture (?)	Massive	Eighth rib. Ether. Pus, 1000 c.c. Second rib resected 1 month later	Recovered	Over 9 months	
6	Elias R., 3 years July 29, 1909	L	5 weeks +	Metapneumonic, culture (?)	Massive—"Necessitatis:"	Sixth rib. Ether, 500 c.c. creamy pus	Recovered	?	Not traced.
7	Sam. S., 5 years July 29, 1909	L	11 days	Metapneumonic, mixed (colon)	Encapsulated, parietal	Eighth rib. Ether. Pus, thin and turbid; colon small	Recovered	?	Not traced.
8	Wm. L., 50 years Sept. 14, 1912	R	2 months +	Metapneumonic	Encapsulated, interlobar, discharging through bronchus	Eighth and ninth ribs. Eucain. 30c.c. extremely fetid pus	Died	.....[1].....	Death in 2 days from sepsis.
9	Minnie Y., 59 years Dec. 25, 1912	L	19 days	Metapneumonic, pneumococcus	Encapsulated, parietal	Eleventh rib. Cocain. Semi-solid pus	Died	.....[1].....	Death in 6 days from bed-sores, etc.
10	Mary O'B., 5 years Jan. 22, 1913	L	2 weeks	Metapneumonic, pneumococcus	Massive	Ninth rib. Ether, 200 c.c. yellow, curdy pus	Recovered	8 weeks[1]	
11	James F., 7 years Feb. 14, 1913	L	3½ weeks	Metapneumonic, pneumococcus	Massive	Eleventh rib. Ether 500 c.c. yellow, creamy and curdy pus	Recovered	3½ months	
12	Walter H., 15 years Aug. 26, 1913	L	10 months	Tuberculous. Smear: micrococci Culture: no growth	Massive	Eleventh rib. Ether. Greenish pus	Recovered	4 weeks[1]	
13	Edna P., 7 years Aug. 27, 1913	L	6 weeks	Metapneumonic, diplococcus	Massive	Intercostal incision, 6th interspace. Eighth and ninth ribs resected 2 months later; bloody pus	Recovered	4 months after intercostal incision. 8 weeks after rib resection.[1]	
14	Margaret P., 12 years Nov. 1, 1914	L	12 days	Followed peritonitis (appendicitis?), streptococcus	Massive	Eighth rib. Gas. 150 c.c. pus. Later, drainage of intraperitoneal pelvic abscess	Recovered	10 weeks[1]	Streptococcus grown from blood. Also metastic osteomyelitis of phalanx (staphylococci) and furunculosis

15	Hugh McI., 43 years Nov. 8, 1914	R	4 weeks	Metapneumonic, pneumococcus	Encapsulated, parietal	Ninth rib. Eucain	Recovered	?	In good health one year later.
16	Joseph R., 19 years Dec. 4, 1914	L	3 weeks	Metapneumonic, diplococci	Encapsulated, 2 parietal empyemata	Eighth rib. Eucain	Recovered	?	Tube fell into cavity after operation.
17	Gerald L., 28 years Dec. 11, 1914	L	3 weeks +	Tuberculous for 7 years. Streptococcus	Massive, *pulsating empyema necessitatis*	Ninth rib. Eucain. Prune-juice pus smelling of hydrogen sulphide	Recovered	?	Not traced.
18	Tony V., 2 ars Nov. 23, 1915	R	5 weeks +	Metapneumonic, streptococcus and staphylococcus	Encapsulated, inter-lobar	Seventh rib. Ether	Recovered	10 weeks [1]	In good health 1 year later.
19	Joseph F., 9 years Nov. 30 1915	R	17 days	Metapneumonic, mixed (colon)	Massive	Ninth rib. Gas. 500 c.c. colon smelling pus	Recovered	9 weeks	In good health 9 months later.
20	Francis P., 11 years Dec. 3 1915	R	2½ weeks	Metapneumonic, streptococci, diplococci, staphylococci	Massive	Seventh rib. Ether. 200 c.c. pus. 7 weeks later, resection of sixth and seventh ribs, 150 c.c. thin fetid pus (streptococci only)	Recovered	5 months (3 months after second operation)	In good health 1 year later.
21	Joseph B., 34 years Dec 10, 1915	L	3 weeks +	Metapneumonic, staphylococcus	Encapsulated, parietal, *empyema necessitatis* with independent pleural effusion	Eleventh rib. Novocain. Turbid fluid, 300 c.c. Intercostal incision, seventh interspace, thick yellow inodorous pus	Recovered	7 weeks [1]	In good health 9 months later.
22	Edward S., 7 years Dec. 11, 1915	R	3 weeks +	Metapneumonic, staphylococcus	Massive	Eighth rib. Gas	Died	[1]	Death 1½ days from noma of mouth.
23	James C., 3 years Jan. 7, 1916	L	3 weeks +	Metapneumonic, pneumococcus	Massive	Tenth rib. Ether. 250 c.c. creamy pus	Recovered	9 weeks [1]	Measles during convalescence. In good health 7 months later.
24	Philip G., 14 years Jan. 14, 1916	L	4 weeks	Metapneumonic, streptococcus	Encapsulated between lung and diaphragm	Tenth rib. Novocain and gas. 500 c.c. pus	Recovered	7 months	Streptococcus from blood. In good health 4 years later.
25	Wm. B., 30 years Jan. 14, 1916	R	9 weeks +	Metapneumonic, pneumococcus	Encapsulated, inter-lobar	Ninth rib	Recovered	3 weeks [1]	Not traced.
26	Kate D., 38 years Aug. 5, 1916	L	6 weeks +	Metapneumonic	Encapsulated, inter-lobar, against pericardium	Seventh and eighth ribs. Novocain	Died	[1]	Death 8 days, unrelieved. Pus not found at operation.
27	James McG., 30 years Nov. 17, 1916	L	3 weeks	Metapneumonic, smear and culture, negative	Encapsulated, between lung and diaphragm	Ninth and tenth ribs. Eucain	Recovered	5 weeks [1]	In good health 3½ years later
28	Wm. B., 20 years Nov. 17, 1916	L	4 weeks	Metapneumonic	Encapsulated, parietal and inter-lobar	Fourth and fifth ribs. Eucain	Recovered	Over 4 months	In good health 3½ years later.
29	John L., 23 years Dec. 29, 1916	L	3½ weeks	Metapneumonic, pnemococcus	Encapsulated, two separate empyemata, both parietal	Ninth rib. Eucain	Recovered	7 weeks [1]	In good health 3 years later.
30	Stanley K., 9 years Jan. 16, 1917	R	2 weeks +	Metapneumonic, diplococcus	Massive	Eleventh rib. Eucain. Reddish-brown pus	Died		

[1] Patients who continued under writer's supervision until death or healing of sinus.

ANALYSIS OF 43 OPERATIONS FOR EMPYEMA—*Continued*

No.	Name, age, date of operation	Side affected	Duration of illness before operation, including a preceding pneumonia	Metapneumonic or Tuberculous; and *Culture*	Massive or Encapsulated	Operation and Anæsthetic	Result	Sinus healed	Remarks
31	Robt. Smith, 7 years Jan. 18, 1917	L	3 weeks +	Metapneumonic, negative	Massive	Eleventh rib. Eucain. Reddish-brown pus	Died		
32	Whitman L., 46 years Jan. 24, 1917	R	3 weeks +	Metapneumonic, diplobacillus	Massive	Eleventh rib. Eucain. 250 c.c. creamy inodorous pus	Recovered	7 weeks	In good health 3 years later.
33	John B., 26 years Jan. 24, 1917	R	2 weeks +	Metapneumonic, pneumococcus	Massive	Eleventh rib. Eucain. 1000 c.c. curdy yellow pus	Recovered	Not traced	Desperately ill before operation (jaundiced).
34	Wm. D., 16 years Jan. 26, 1917	R	3 weeks	Metapneumonic, pneumococcus	Massive	Eleventh rib. Eucain. 500 c.c. creamy yellow pus	Recovered	6 weeks[1]	Desperately ill before operation (bilateral pleuritis, endocarditis, and pericarditis). In good health 3 years later.
35	Edward H., 32 years April 8, 1917	R	10 weeks +	Tuberculous-metapneumonic diplococcus	Encapsulated, two distinct empyemata; (1) between lung and diaphragm; (2) parietal	Ninth rib. Eucain.	Recovered	7 weeks[1]	No lung symptoms 3 years later. (Lives in Colorado.)
36	Oliver C., 21 years July 26, 1919	R	3 weeks	Metapneumonic, pneumococcus	Encapsulated between lung and diaphragm	Tenth rib. Novocain. 150 c.c. creamy yellow pus	Recovered	12 weeks[1]	In good health 8 months later.
37	Isadore M. 11 years Aug. 18, 1919	R	19 days	Metapneumonic, pneumococcus	Massive—"Hourglass empyema"	Tenth rib. Novocain. 1000 c.c.	Recovered	3½ weeks[1]	In good health 7 months later.
38	Evelyn S., 19 years Nov. 18, 1919	R	3 weeks	Metapneumonic, streptococci	Massive—"Hourglass empyema necessitatis"	Ninth rib. Novocain. 250 c.c. yellow, creamy malodorous pus	Recovered	8 weeks[1]	Desperately ill before operation. Metastatic abscess under L. scapula during convalescence. In good health 6 months later.
39	Gertrude W., 4 years Dec. 6, 1919	R	4 weeks	Metapneumonic, diplococci and staphylococci	Massive	Ninth rib. Novocain. 100 c.c. pus	Recovered	6 weeks[1]	In good health 4 months later.
40	Thos. W., 16 months Jan. 9, 1920	L	3 weeks	Metapneumonic, pneumococci	Massive	Tenth rib. Novocain. Thin serous pus.	Recovered	13 weeks	
41	Alice M., 29 years Feb. 20, 1920	R	3 weeks	Metapneumonic, pneumococci	Massive	Eleventh rib. Novocain. 500 c.c. creamy yellow pus	Recovered	7½ weeks[1]	
42	Theresa D., 27 years Feb. 20, 1920	L	17 days	Metapneumonic, pneumococci	Massive	Tenth rib. Novocain. 500 c.c. thin turbid flaky pus	Recovered	7½ weeks[1]	
43	James S., 34 years April 9, 1920	L	8 weeks	Metapneumonic, pneumococci	Massive—"Hourglass empyema"	Tenth rib. Novocain. 1000 c.c. greenish-yellow creamy pus.	Recovered		

[1]Patients who continued under writer's supervision until death or healing of sinus.

it, thin turbid pus discharges, with some flakes of lymph, nearly 500 c.c. Finger introduced feels expanding lung, not bound down by adhesions. Large tube drain.

March 5, 1920: Tube replaced by smaller and shorter. Temperature normal ever since operation.

March 19, 1920: Up and about ward. Very little discharge.

April 10, 1920: Tube removed.

April 14, 1920: Went home, wound healed.

CASE XLII.—Empyema, left. Recovered. James S., aged thirty-four years.

April 7, 1920: Admitted to the Episcopal Hospital, Doctor Hopkins's service. Onset February 6th, with influenza, followed by pneumonia with crisis in eight days. Did not get well. Thought by family physician to have tuberculosis. Two aspirations at home four weeks ago in posterior axillary line, left side, were negative.

April 8, 1920: Aspiration showed thick creamy pus. Aspirated in seventh interspace, posterior axillary line. Transferred to surgical service (Doctor Ashhurst).

April 9, 1920: Operation (Doctor Ashhurst)—novocain. Resection of 4 cm. of tenth rib in scapular line. Pleura thickened. Pus spurted 2 metres on opening pleura, creamy, greenish yellow pus. About 2 litres removed. Exudate surrounding lung on costal and diaphragmatic surface (Fig. 22). Tube inserted and anchored with silkworm-gut.

April 23, 1920: Tube shortened.

April 30, 1920: Smaller tube inserted.

May 12, 1920: Tube removed.

May 15, 1920: Went home; granulating wound. No discharge.

June 14, 1920: Wound healed.

OPERATION FOR EMPYEMA IN YOUNG ADULTS *

By Frank E. Bunts, M.D.
of Cleveland, Ohio

The great prevalence of pneumonia in many of the United States cantonments and the resulting incidence of empyema, reaching as high as 10 per cent. in the base hospital to which I was assigned, brought to the surgical service about 175 cases for operation. I am not able at present to give the exact data, but certain clinical facts connected with this series of cases seem to be of special interest.

All cases naturally occurred in young adults of army registration age, and while this may have no bearing upon the results, yet it must be granted that they should by the mere fact of their youth and of their having been admitted to service after elaborate physical examinations, be presumed to be better risks than the average case in civil practice, and to avoid the possibility of inherent differences in the varied cases found in civil practice, I have designated them in a group as young adults.

I. *Classification.*—In concurrence with the Chief of the Medical Service, patients with fluid in the chest were divided into three classes:

(*a*) Those with clear fluid and no microörganisms present.

(*b*) Those with slightly turbid fluid and various bacteria—staphylococcus, colon bacillus, or streptococcus hæmolyticus—present.

(*c*) Those with frank yellow pus and with bacteria present.

II. *Recognition, Diagnosis.*—Physical examination often failed to reveal the location of the accumulated fluid, though its presence was practically certain. The use of the aspirating needle was freely resorted to and a considerable number of punctures were frequently made without successfully locating the fluid. This was due, to a considerable extent, to the fact that many of the accumulations were encapsulated, and particularly to the frequency of interlobar collections of pus.

To obviate the frequent and not always innocuous punctures, early use of the X-ray examination was made in cases able to endure such examination. This greatly simplified and rendered comparatively easy the detection of the location of the empyema, and where stereoscopic plates could be made, immensely facilitated operative procedures.

III. *Treatment.*—Class (*a*). These were aspirated by the physician in charge, or upon request, by one of the surgical staff, but were not transferred to the surgical service.

Class (*b*). These were transferred to the surgical service, aspirated under primary ether anæsthesia, and injected with a small amount of glycerine and formalin (2 per cent. solution). They were watched carefully from day to day, and a failure to show improvement or an increase in the

* Read before the American Surgical Association, May 3, 1920.

severity of their symptoms was followed by immediate operation. In the neighborhood of 16 cases were treated in this manner, of which six or seven recovered without operation. Inasmuch as an occasional case under this classification recovered in which aspiration without the injection of formalin was carried out, it is impossible to say that the latter was a curative agent in the others. All that can be said is that a number got well under its use, and that it did not seem to do any harm in any instance.

Class (*c*). These were all operated upon within twelve hours after their detection, except, and this I believe to be of importance, *those cases where the high fever and physical signs showed the pneumonia to be still active or invading the opposite side.* In these the fluid was aspirated, sometimes repeatedly, until the pneumonic symptoms abated, and then aspiration was proceeded with. It was a self-evident fact in these cases that the empyema was not the determining factor in the critical condition in which these patients found themselves, and a radical operation, or indeed, any operation other than aspiration, would materially lessen thir chance of recovery. This we found to be true, from serious clinical experience.

Anæsthetic.—I have read and heard much discussion regarding the proper anæsthetic to be used in these cases, some saying that only local anæsthesia should be used, and almost all condemning the use of ether. In a few of the very worst cases when it seemed from the greatly debilitated condition of the patient that no general anæsthetic would be tolerated, I used local anæsthesia alone, but in all the others primary ether anæsthesia was used, and, I believe, without any ill effects whatever; the patient being wide awake at the end of the operation. I have not seen severe coughing or respiratory embarrassments result from the proper administration of ether, and the temporary and transient loss of consciousness, I believe, to be an act of mercy to the patient.

Method of Operating.—Local anæsthesia over area of rib to be resected, followed by light primary anæsthesia and rapid incision and excision of bone, insertion of a long ¾-inch rubber drainage tube which was sutured into the wound, tight suturing of the wound about it and a clamp to the tube to prevent escape of fluid.

Subsequent Treatment.—The patient was put in a semi-recumbent position or the head of the bed elevated by blocks, and the tube end inserted into a drainage bottle containing some antiseptic fluid and attached to the side of the bed. The clamp on the tube was opened up for a few moments every half hour, allowing a small amount of fluid to escape until the chest cavity was evacuated and danger of sudden respiratory or circulatory changes eliminated. The clamp was then removed entirely and drainage allowed to continue. At the end of a week or ten days, rarely earlier, the cavity was washed out twice daily with varying solutions, such as iodine, sterile water, normal saline, boracic acid, formalin and glycerine, and at two-hour intervals when the Carrel-Dakin method was used. The large number of cases operated upon gave excellent opportunity to try

out a series with each method. In from one to two weeks, when the discharge had greatly diminished, the large tube was removed and progressively smaller short tubes inserted.

Blowing into a bottle was insisted upon in every case, but not until the *unaffected side had been carefully strapped with adhesive plaster in a manner similar to that used in fractured ribs,* the object being to prevent emphysema of the well lung, if possible, and to hasten the expansion of the collapsed lung.

As soon as the patient was able to be up, light setting-up drills and breathing exercises were instituted. Beds were moved out of the wards on to the porches, foods were administered as frequently and in as great quantity as the patient could take them. Heroin, and occasionally morphine, were given during the first two or three days for pain or cough, threatened œdema of the lungs was combated with digitalis, atropine and oxygen, and in the later stages, iron, usually in the form of the syrup of the iodide, was given.

IV. *Results.*—There was a mortality of approximately 13 per cent. This included every case operated upon, regardless of post-mortem findings. No deaths occurred immediately after operation, two days to three weeks being the subsequent range of life when death followed operation, depending upon complications resulting.

New accumulations of pus, usually interlobar, were sought for and found by the use of the X-ray before using the needle in those cases which did not clear up under treatment, and secondary operations were sometimes necessary for their evacuation.

No case required extensive rib resection (Estlander operation), decortication, or other procedure, for obliterating the chest cavity. Two-thirds of all the recovery cases were either in France with their regiments or temporarily in a convalescent hospital with the wounds healed, and at the time of my detachment from the base hospital there were none who were in a condition which would not admit of their early discharge to duty or to a convalescent hospital without further operation upon the chest.

Complications.—Infective arthritis occurred in a small number of cases. Pneumonia on the opposite side and occasionally empyema on the opposite side made serious and, in one or two instances, fatal complications. Endocarditis and myocarditis were rare occurrences.

Autopsies.—Until countermanded, autopsies were held on all deaths following empyema. The results of these examinations were most illuminating and valuable. *In not one case could we ascribe the death to empyema.* It was invariably found that the drainage, so far as the part attacked was concerned, was satisfactory, but the cause of the death was a pyæmia rather than an empyema, broken-down and suppurating bronchial and mediastinal glands, pus in the pericardium, pultaceous areas in the spleen, abscesses in the liver, multiple abscess in the lungs, and free pus

in the peritoneal cavity, gave evidence of the general rather than the local character of the condition. I was informed by a Chief of Service in one of the cantonments, that these cases were not classified as death from empyema, but rather from pyæmia. If this be a proper disposition of them, then the mortality from simple cases of empyema was practically nil in the series of cases to which this paper refers.

Remarks upon Operations and Treatment.—The results of autopsy findings must be carefully considered before advocating any special line of treatment or operation. Following the great number of operations throughout the United States for empyema, a great array of opinions and operations was advocated and many claims for excellency presented which can scarcely be admitted in view of the fact that except under the most unfavorable circumstances and in the hands of the most inexperienced operators and of totally inadequate technic, deaths do not occur by reason of the method of operative procedure, but by reason of the inherent character of the pyæmia present, which precludes the possibility of efficient surgical intervention. This being the case, I do not present the method of operation outlined in this case as being superior to others, but in view of its low mortality, of the comfort of the patient, of its excellent permanent results, and of the cleanliness and absence of disagreeable odor in a ward full of these usually offensive smelling cases, I believe it to be worthy of consideration as a good method of operation.

THE ETIOLOGY OF CHRONIC EMPYEMA

By Walton Martin, M.D.
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The impression that I have gathered, from studying my own cases of chronic empyema and from talking with other surgeons, is that chronic empyema is a very uncommon outcome of the acute infection of the pleura when the operation for drainage is performed and the after-care supervised by an experienced surgeon. I have talked over this question with men holding most divergent ideas regarding the treatment of acute empyema, and, if they are interested in the subject, they invariably tell me that with their method they never see chronic suppurative pleurisy develop.

This has suggested to me that in a large percentage of these cases the causative factor lies in the treatment of the acute condition. That is, in only a small percentage of cases are there present from the beginning conditions which will invariably lead to chronic empyema. The first thought, on seeing a large, unyielding cavity develop in the pleura following the drainage of an acute empyema, is that since the lung collapsed when the chest was opened it has remained collapsed because an open pneumothorax has been established; positive atmospheric pressure pressed the lung in, therefore, negative pressure must be established and maintained. Every recrudescence of the empyema question is regularly followed by a great many devices for so-called bloodless thoracostomy. Of course, the problem cannot be solved in this way alone. Statistics show, in fact, that more chronic empyemata result from continuous puncture aspiration drainage than from any other form of treatment except repeated aspiration.

I have thought, therefore, it might be of interest and profit to discuss other etiological factors, drawing my conclusions from a series of twenty-four cases on which I found it necessary either to do an extensive thoracoplasty or decortication.

The very definition of the condition is a debatable point. Is the persistence of pleural suppuration for two months, four months or six months indispensable? Is the ease with which it can be cured or the readiness with which the cavity can be disinfected the essential? One usually means, I think, by chronic empyema a persistent collection of pus in the pleural cavity with little or no tendency to heal; that is, an empyema in which, after the evacuation of the pus, the lung does not expand again and an infected cavity remains. These unfavorable anatomical conditions of the lung, pleura, and thoracic wall are reached much faster in some cases than in others, so that a definite time limit cannot be given, nor is there a definite pathological picture. There is obviously a gradual transition, with many intermediate forms, between the acute and chronic condition, but the essential feature is an infected cavity

with rigid walls. If a collection of pus is not opened for several months or remains untreated until it bursts through the chest wall or lung, the pulmonary and visceral pleura, in response to the repeated bacterial irritation, become thickened and unyielding. The lung, compressed by the new-formed granulation tissue, is firmly held in a more or less collapsed condition. The pleura is converted into a thick layer of organized connective tissue. The deeper layers, those in contact with the lung, after a varying time become so well organized that they will not disappear even after the bacterial irritation ceases by giving free exit to the pus. This pleural response to irritation is probably dependent on the type and virulence of the bacteria and the special individual resistance of the pleura. However, there is a direct time relation between the duration of the infective process and these changes in the pulmonary and parietal plura, and, although there is a great variation in this time relation, yet it holds true that the ability of the lungs to expand has a direct relation with the interval between the onset of the empyema and the free evacuation of pus. If the pus be retained, for example, until it bursts through the chest wall a chronic empyema invariably results.

The thickening of the pleura in response to bacterial irritation and the binding down of the compressed lung, so that when the chest is opened the lung no longer expands, is the pathological picture of chronic empyema. The inner surface of the suppurating pleural cavity is irregular and covered with pus and detritus. Its outer surface is made up of fibrillar connective tissue. It is not materially different from the walls of an abscess elsewhere in the body and obeys the same laws, so that the thickness and rigidity of the wall bears not only a definite relation to the duration of the infection, but to the amount of pressure or tension of the exudate set up by the bacterial irritation. Every time there is an increase in tissue tension about a nidus of pyogenic infection there is a local response manifested by an increase in the surrounding new-formed tissue walling it in. If the infected pleural cavity be opened and free passage be given to the exudate there will be found a direct relation between the adequacy of the drainage and the reaction and consequent thickening of the pleura. If intermittent or inadequate drainage be established the infection will not terminate, the pleura will continue to react to the bacterial irritation, and the granulation tissue forming the wall of the pleural cavity will become better and better organized. Now the conditions in the thorax are such that insufficient drainage is very liable to occur. Aside from the position of the original incision, whether a rib be resected or there be an intercostal incision, whether large drainage tubes be introduced or an attempt be made to make an air-tight joint about a tube, there is a marked tendency, as soon as wound healing begins, to close the drainage tract. The shifting planes of the thoracic wall and the crowding together of the ribs tend very quickly to break the continuity of the drainage path. The soft parts heal about the tube, and,

although it may be patent, it is often sufficiently compressed to interfere with the absolutely free exit of the pus which is essential to lung expansion.

In Case XXI the patient presents an example of chronic empyema resulting from inadequate drainage. One month after the first aspiration a drainage tube was inserted. At the end of five months he still had a small tube in the chest, the soft parts were contracted about the tube, the rib had regenerated, forming a bone ring about the orifice in the soft parts. Every few days he had a rise of temperature; he had lost his appetite; he was anæmic; the muscles of his arm and side were atrophic; his chest contracted. In short, he presented the usual picture of chronic suppurative pleurisy. The retention of pus and the imperfect drainage could readily be demonstrated by injections of a weak solution of methylene blue. Notwithstanding coughing and straining, greenish pus was still pouring out of the wound three days later and the urine was stained for several days, showing the absorption even through a thickened pleura. I know of nothing more surprising than to see a patient four or five weeks after an operation for empyema, with a small drainage tube still in the chest, with the soft parts healed firmly about it, with abundant discharge of pus, but with an afternoon temperature, a rapid pulse and no appetite, change after reoperating and resecting a portion of a rib and establishing free drainage. The temperature becomes normal, the appetite returns, the lung often expands in a surprisingly short period. A similar but slower result followed free drainage and sterilization of the cavity in this patient.

There is yet another outcome of a focus of infection, uncommon to be sure, but of great interest. The exudate does not break through the surrounding wall nor is an external passage provided for it. The circumscribing wall becomes very greatly thickened, the virulence of the bacteria becomes more and more attenuated; finally, a static condition is reached and the lesion is no longer active. It remains for months and years latent. A most interesting problem is here presented regarding the life-history and metabolism of non-spore bearing pyogenic bacteria in one of these chronic abscesses. The examples of this condition that we are familiar with are the chronic pyogenic bone abscesses. From trauma or some obscure cause the delicate balance between the living cells of the abscess wall and the microörganisms may be at any time upset and the bacteria again begin to grow; tissue tension is again present, with its local and constitutional signs months and years, even, after the original infection. There are records of circumscribed collections of pus in the pleural cavity that have apparently been there for years and that were discovered accidentally at autopsy. Brin reported the autopsy of a woman, aged sixty-four years, operated on for stenosis of the common duct. The woman died on the fifth day. In the left pleural cavity there was an abscess filled with thick yellowish pus and broken-down

material. It was situated between the diaphragm and the under surface of the lung. The surrounding pleura was 1½ cm. thick and the costal pleura calcareous. There was no communication with a bronchus. There was no record in the history of the patient of an illness other than that of the biliary colic.

A far commoner condition is the latent collection of pus following operation for acute empyema. A patient has an acute empyema; the pleural cavity is opened and drained. After a time the temperature is normal, the discharge diminishes or almost ceases, the drains are removed, and the external wound heals. The patient is discharged from a hospital service. He, however, after his return home never feels really well. His appetite is poor; from time to time he feels feverish; he has a slight cough. The physical signs over the affected side do not return to normal; the Röntgen ray may be indistinct and may only show great thickening of the pleura. One of my patients (Case XIX) with this condition was sent to Loomis Sanitarium for tuberculosis. He had had pneumonia and an operation for empyema six months previous at his own home. He remained at the sanitarium for four months, although no tubercle bacilli were found in the sputum. He then began to run a high temperature. An exploring needle was inserted into his chest near the old empyema scar and pus withdrawn. At operation there was a very large rigid-walled cavity involving the whole right pleura. In another (Case XI), a young Italian girl, there was an operation for empyema in December, 1913. After several months the sinus closed. She returned home, but never regained her health. She was readmitted two years later with a high temperature. An exploring needle inserted near the old scar withdrew pus. At operation there was a large empyema cavity, the lung was compressed against the vertebral column, the parietal and visceral pleura were enormously thickened.

Foreign bodies, usually drainage tubes, have been frequently found in the pleural cavity. It is usually taken for granted that they are responsible for the chronic suppurative pleurisy which always accompanies their presence. I have operated on two patients with this condition (Cases IV and VII). In one of them, a man, aged thirty-eight years, two tubes were removed. He had had a persistent sinus and chronic empyema for four years. The patient usually leaves a hospital with a draining sinus and a drainage tube in place. At one of his dressings no tube is found. From the fact that the sinus is narrow and that he has no realization of the cavern within, the doctor dressing the patient when the tube is lost assumes that the tube has fallen out, not in. He usually inserts a new tube. In other words, drainage tubes readily fall into chronic empyema cavities rather than cause them. Their presence there is an added factor, of course, in causing the persistence of the suppuration.

In nearly every series of chronic empyemata reported a certain number are caused by tuberculosis. A distinction should be made between

tuberculosis of the pleura, secondary tuberculosis developing in a patient suffering from chronic suppurative pleurisy and suppurative pleurisy occurring in a patient suffering from tuberculosis. In my series there were three cases of tuberculous pleurisy. In two of these there was an associated pyogenic infection.

In 12 recurrences following acute empyema at the site of the primary operation, Stevens found the streptococcus hæmolyticus (the organism that caused the original infection) in 11; 1 was sterile. In 9 recurrences distant from the sinus, including undiscovered pus pockets from multilocular empyema, 4 were due to the streptococcus, 3 were sterile, and 2 contained staphylococcus.

But the effort to base the prognosis and treatment on the type of infection made by Netter has not proved satisfactory. The infection is frequently mixed and there are extraordinary differences in virulence in different epidemics in both the pneumococcus and the streptococcus. The staphylococcus is rarely found unassociated with one or the other pyogenic microörganisms. Its presence in pure culture led Netter on two occasions to predict tuberculosis and in each instance his prediction proved true. The tendency of both the streptococcus and staphylococcus to persist in the tissue and the phenomena of reinfection and secondary infection with these organisms is significant. In making routine bacteriological examinations of granulating wounds one is struck with the ease with which reinfection takes place from some cause often difficult to discover: a piece of necrotic tissue, a minute foreign body or a small pocket with retained exudate. The wound surface which has been almost free from bacteria is suddenly swarming with them again. When the same type of bacteria start to grow luxuriantly we speak of reinfection; when a new organism is added we speak of secondary infection. We are all familiar with the effect of secondary infection in tuberculosis. Kiener reports two interesting observations of secondary infection following empyema: one, a staphylococcus infection, was grafted on a streptococcus; in another a staphylococcus was added to a tuberculous infection. In the subsequent persistent infection the staphylococcus was the prevailing organism.

In Cases II, VIII and XI, I have been able to follow, to a certain extent, the sequence of events in the development of the chronic empyema. In Case II, for example, at operation the lung was found compressed against the vertebral column; the cavity was very large, involving the whole chest. It was partially filled with foul-smelling pus; the pleura was greatly thickened. The patient was eighteen years old. He had been in good health before the onset of the acute empyema, three years before. The diagnosis of pus in the chest had been made early and an operation performed and the drainage tube inserted. After ten days this tube was replaced by another connected to a rubber suction bag and an effort had been made to make an air-tight joint between the thoracic wall and the

drainage tube by iodoform packing, perforated rubber dam, and adhesive plaster. After several weeks the patient was allowed up and permitted to walk. The drainage tube was still in the chest and the suction bag still attached. He was operated on in May. In July he still had a tube in the chest. During this month the tube was removed and the external wound closed. He went home but continued in poor health. He returned to the hospital nine months later for cough and persistent temperature. The chest was opened, a piece of rib was resected, and a drainage tube inserted. The pus evacuated was brownish and very foul smelling. Again the suction bag was applied. There was the same sequence of events; he returned home, the wound again closed, but he never regained his health, although he was not actually sick. He had slight fever, persistent cough, and shortness of breath. In the patient referred to the exudate at the second operation was foul smelling. The original pyogenic infection had become secondarily infected by putrefactive organisms.

This question of secondary infection and reinfection and its relation to chronic empyema has been little studied. Of course, it presents unusual difficulties. Secondary surface contamination is almost certainly present in every granulation wound or cavity lined with granulations that communicates with the external air. This by no means necessarily denotes secondary infection. Forty years ago incision and drainage of the chest was unsatisfactory. It was soon generally recognized that the patients were reinfected after the operation. When both the operation and the after-treatment were carried out under antiseptic precautions there was an astonishing improvement in results. The study of the statistics between 1875 and 1895 is very instructive. I believe these cases I have referred to are examples of chronic empyema resulting from repeated reinfection of the pleura and intermittent drainage. A drainage tube is a foreign body. When a tube is retained in any wound until healing takes place it is soon compressed by contracting healing tissue. The pressure is finally sufficient to interfere with the nutrition of the granulations in contact with the tube; surface necrosis results. A condition of repair and interference with repair is established. The trauma is added to by the movement of the tube in the drainage tract; the greater the activity of the patient the more this displacement of the tube is likely to occur. By these repeated injuries of the granulations the protecting lining wall of round-cells is broken again and again. Surface organisms, usually the staphylococcus or streptococcus, penetrate the tissue and infection, not contamination, is established. If the tube be long the walls of the drainage tract are made up not only of thoracic wall, but the pleura. The exudate, made up of leucocytes and microörganisms, oozes between the tube and the granulating wall embracing the tube. Some of it trickles into the space still present in the pleura, is shut off by the granulations and imprisoned. There is a renewed intrapleural exudate sufficient to hinder expansion of the lung. When the tension of the exudate becomes sufficient it again

escapes externally. One of the most admirable principles of the Carrel treatment is the scrupulous avoidance of secondary infection and damage to the granulations, and it seems to me, as soon as the healing wound has contracted so tightly about a drainage tube that it is inserted with difficulty, it is better to remove the tube or reoperate.

The influence of the anatomical arrangements of the adhesions between the pulmonary and parietal pleura at the time of the original infection has been suggested by Homan as an etiological factor of importance in determining the tendency of chemistry. If the lower margin of the lung be held down by adhesions to the diaphragm he believes that the prognosis is favorable; the lung will expand readily after drainage. If, on the other hand, the lung is collapsed and adherent and the lower margin is not in contact with the diaphragm, the prognosis is bad; chronic empyema is likely to occur. He shows a silhouette taken from a Röntgen-ray plate in one case of chronic empyema which represents one of the types seen in chronic suppurative pleurisy when there has been a large collection of pus in the chest. The lung is usually either compressed so that there is only a narrow band of lung tissue in contact with the vertebral column or, as in his illustration, drawn upward away from the diaphragm. The Röntgen-ray photographs in Cases VIII and XX are very like his diagram. Obviously a small sacculated empyema favorably situated for drainage offers a good prognosis. Also, obviously, a chronic empyema involving the entire pleural cavity, with lung completely collapsed, presents a more difficult problem than a circumscribed chronic empyema. His theory seems to me, however, to attribute a permanency to the original plastic adhesions, as if they were unmodified by the duration, character, and treatment of the infection, unaltered by protophytic ferment and proteolytic antiseptic solutions, uninfluenced by the mechanical factors of intra- and extrapulmonary pressure. A similar theory applied to the permanency of the original plastic adhesions about a focus of infection in the peritoneum would lead to conclusions at variance with common experience.

The shape of the empyema cavity has a significance in determining the readiness with which it can be drained and cured. If it is irregular, with secondary pockets or diverticuli, or presents a considerable cavity connected with the main cavity by a narrow channel, all the phenomena of intermittent drainage or incomplete drainage may be produced by the shutting off of a loculus or by the more or less complete blocking of a channel; drainage of the main pocket will evidently be inefficient. Multilocular empyemata and lung abscesses are frequent autopsy findings. In fact, the mortality in acute empyema seems to be largely due either to the fact that the empyema is only one of the localizations of a general infection which may lead to death with lesions in the lungs, pericardium, and meninges, or to undrained, circumscribed collections of pus. For example, in the necropsy findings reported in 16 fatal cases at Camp Sheri-

dan, Ohio, 14 had one or more undrained collections of pus. In a fifteenth there was a miliary tuberculosis. In Case II, for example, an abscess was found in the left chest above the fourth rib containing 200 c.c. of pus. The pleural cavity below that point had been drained. There was a second pus pocket between the upper and lower lobe. If death does not occur and the pus from one of these secondary foci finds its way into the main cavity we have produced an irregular cavity imperfectly drained. In some of these cases a secondary opening occurs spontaneously through the lung, either a lung abscess breaking into the pleural or a loculated collection of pus finding its way through the lung and draining through a bronchus. Chronic empyema results when the microörganisms are attenuated and there is inadequate and intermittent drainage. Obviously, the virulence of the infection or the complete absence of drainage leads either to the death of the patient or the recognition and early treatment of the condition.

In Cases IV, V, VI, VII and IX the large empyema cavity was draining both through the chest and through the lung. In Case IX there was an abscess in the upper lobe that communicated with a bronchus and with the pleura, so that there was a dumbbell-shaped cavity draining at each end. In the case of Romain there was a similar condition, but the abscess was in the lower lobe. In these cases the large opening into a bronchus is an etiological factor in the persistence of the suppuration process. At times there is free drainage through the bronchus, at times it is blocked and the pus from the infected area in the lung backs up and empties into the pleural cavity. We have again produced the phenomena caused by intermittent collection, an exudate under tension about a focus of infection.

I believe, then, that the cause of the chronicity does not lie in any one factor, such as the variety of the organisms, the type of the exudate, the nature of the infection, or the anatomical arrangements of the original adhesions, but in the complex made up of all these causes, the underlying element being repeated infection and reinfection of the pleura, causing its thickening and rigidity and the binding down of the lung. We have abundant evidence from autopsy findings made within the first week of drainage in extensive infection of the pleura, from Röntgen-ray examination and from the physical signs that the lung will expand readily and quickly in favorable cases within the first week if there is a free passage made for the exudate, and this expansion occurs even under the ordinary aseptic or antiseptic dressing. From the figures of Cestan, in 1208 cases 1.8 per cent. were not completely cured after simple pleurotomy and drainage, whereas with aspiration drainage after incision the failures were 3 per cent., and after puncture drainage with aspiration the failures were 6 per cent. Stevens reported in 100 cases treated by simple incision that 56 healed satisfactorily, 34 healed after surface sterilization of the cavity with Dakin's solution; in 10 a thoracoplasty of moderate extent was

indicated. In 23 cases treated by drainage and irrigation with Dakin's solution from the beginning there were 3 thoracoplasties for foci distant from the sinus. There were no recurrences at the site of the original operation.

Statistics, taking no account of the experience of the operator and the supervision of after-care of the patient or the original complications, may readily be misleading.

It was long ago pointed out by Schede that a closed pneumothorax, uninfected, rapidly disappeared. During the war Bastianelli found satisfactory restoration of lung expansion after an artificial pneumothorax which had been established and maintained for from twelve to fifteen days. Since we have made routine Röntgen-ray examinations I think many of us have been surprised when evidences of infection are no longer present, as shown by the mucoid nature of the wound secretion or by negative bacterial findings, to see that the lung expands satisfactorily if the outer wound is allowed to close, even in the presence of a considerable pneumothorax. When expansion has taken place and the infection has ceased, Röntgen-ray examination shows that the thickened pleura gradually returns to a layer so thin that it is no longer opaque to the Röntgen ray even when there has been long-continued infection and the pleura has been seen to be much thickened at operation (Case II).

I believe that chronic suppurative pleurisy, excepting when there are complicating conditions in the lung or an underlying tuberculous infection, should not occur. I feel sure that in the majority of cases there has been a failure to appreciate the fundamental principle involved in treating an infected cavity; that an absolutely free external passage for the exudate must be provided as long as infection is present; that scrupulous cleanliness must be observed in the after-care; that one must be always on the lookout for the possibility of secondary loculi and foci of infection and shut-off portions of the drainage tract; that in a widely drained cavity surface contamination does not lead necessarily to reinfection, but that in a poorly drained cavity reinfection and secondary infection are very liable to occur. I believe that surgeons with very different ways of operating and very diverging ideas regarding after-care get equally good results provided these essentials are borne in mind: That the entrance of air through the thoracic wound to any desirable degree can be assured by very simple means; that although the danger of early operation in streptococcus infection while pneumonia is still present has been recently generally recognized, this by no means implies the desirability of a long delay and treatment by aspiration after the pneumonic process is over and the exudate is purulent or seropurulent in character. I think no case should be allowed to leave a hospital with a drainage tube in the chest; that all patients with a closed, even if apparently sterile, pneumothorax should be kept under close observation until it disappears. I think that there should be a more general recognition that not only high temperature and obvious signs of infection indicate a pent-up collection of pus,

but that a slight afternoon temperature, a rapid pulse, loss of appetite, vague discomfort in the chest—in short, a failure to return to vigorous health—are very suggestive of a hibernating pleural abscess. Finally, that in the treatment of acute empyema will generally be found the etiological factors of the chronic condition.

In over one-half of the cases of chronic empyema in my series there was no complicating lesion, no infection with tubercle bacilli, no complexity of the original cavity.

OBSERVATIONS ON THE TREATMENT OF CHRONIC EMPYEMA

By George J. Heuer, M.D.
of Baltimore, Md.

In 1913 a patient who had been treated for pulmonary tuberculosis by artificial pneumothorax came to us with a tuberculous empyema. We resected a rib and drained an empyemic cavity containing over 1000 c.c. of thick greenish pus in which were myriads of tubercle bacilli. The lung, due to its long period of collapse, was incapable of expansion and the patient's condition did not permit a thoracoplastic operation. Under the circumstances, we tried repeated injections of Beck's bismuth paste, and were astonished at the result. The tubercle bacilli disappeared from the discharge, the discharge itself became a clear straw-colored sterile fluid, and the sinus tract spontaneously closed. The intrapleural cavity never became obliterated, yet the patient went about not at all inconvenienced by its presence (Fig. 1). So far as we can gather, this is the first case in which the complete sterilization of a chronic empyemic cavity followed the prolonged use of antiseptic substances. It demonstrated to us that it was possible to sterilize empyemic cavities and that sterile intrapleural cavities need not be surgically obliterated, but were quite compatible with good health.

Since that time we have been deeply interested in the treatment of chronic empyema. Our experience soon showed that we, at least, were not able within a reasonable time to sterilize all empyemic cavities by the injection into them of bismuth paste; and, guided largely by animal experimentation, we have therefore sought for other methods which would cure chronic empyema and still avoid the mortality and the mutilation of thoracoplastic procedures such as the Estlander and Schede operations. Our observations on the treatment of chronic empyema before and since the discovery of the Carrel-Dakin method of treating infected wounds, concern the description of and the results obtained by these methods.

1. *Bismuth Sterilization of Empyemic Cavities.*—In addition to the case described in our introductory remarks, we have attempted the sterilization of chronic cavities with bismuth paste in six cases. In five of them a preliminary rib resection to establish a satisfactory sinus was necessary. The method is now well known and we need not pause over it. We should, however, like to add our experience to that of others who have had favorable results. In one patient with a large tuberculous empyemic cavity similar to the one described, we failed to completely sterilize the cavity during the period (two months) he was under our care; yet there was marked improvement, as indicated by the fall in the temperature and the gain in weight. In the smaller empyemic cavities, however, and

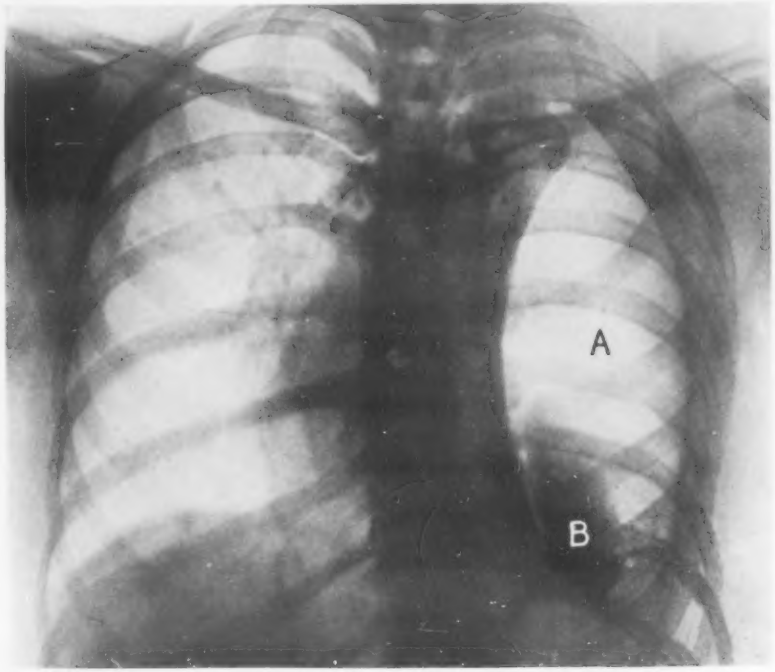

FIG. 1.—A tuberculous empyemic cavity sterilized by bismuth paste. *A*, empyemic cavity; *B*, a small mass of bismuth paste remaining in the cavity at the time of the closure of the sinus.

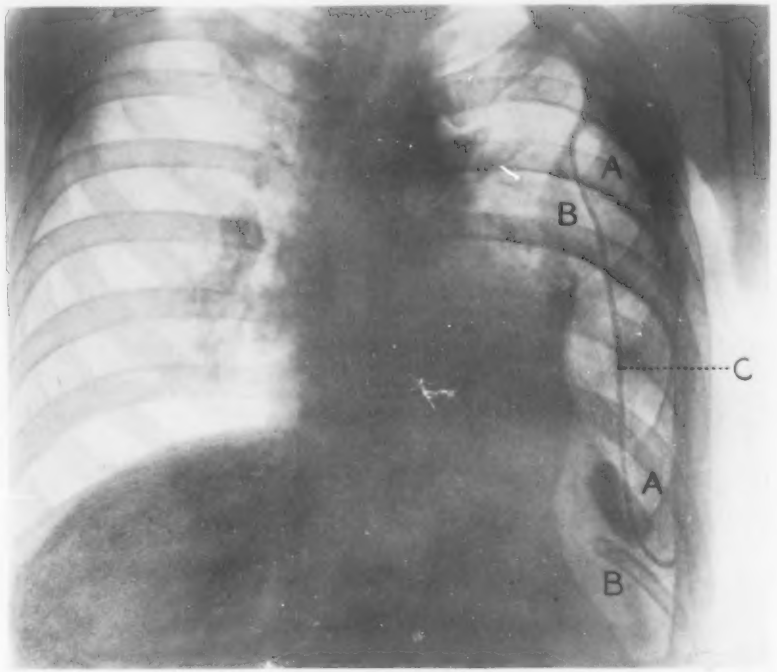

FIG. 2.—A tuberculous empyemic cavity in which an attempt was made to substitute a sterile extrapleural cavity for a septic, intrapleural cavity by stripping the parietal pleura. (See text, Method 2.) The result, due to the thickness and rigidity of the parietal pleura, was a partial and not a total obliteration of the intrapleural cavity. *A*, extrapleural cavity; *B*, intrapleural cavity; *C*, the mobilized parietal pleura. (X-ray taken about ten days after operation.)

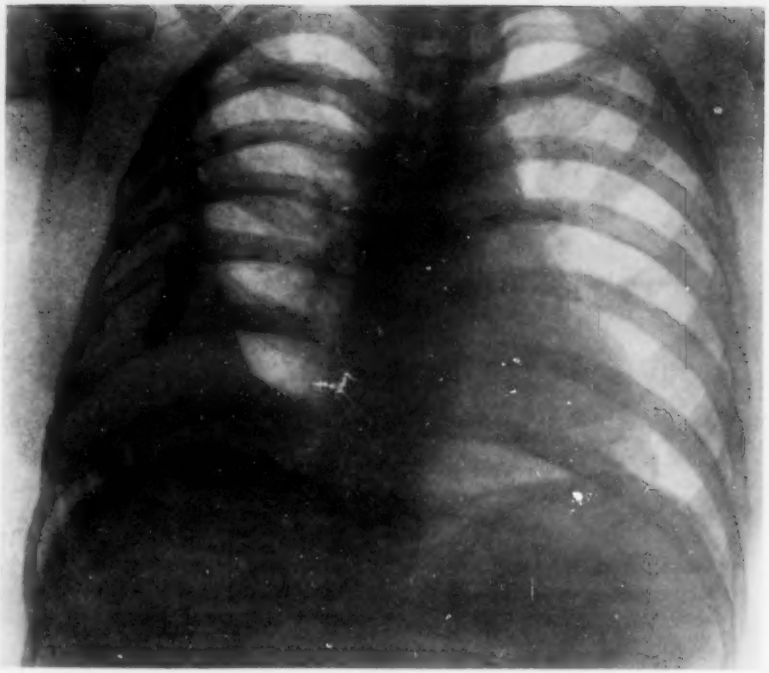

FIG. 3.—A chronic empyemic cavity treated by mobilization and excision of the parietal pleural followed by irrigations of the extrapleural cavity with Dakin's solution. (See text, Method 3.) The small dense shadow is the remains of the bismuth paste injected to determine the form and position of the cavity. (X-ray taken before operation.)

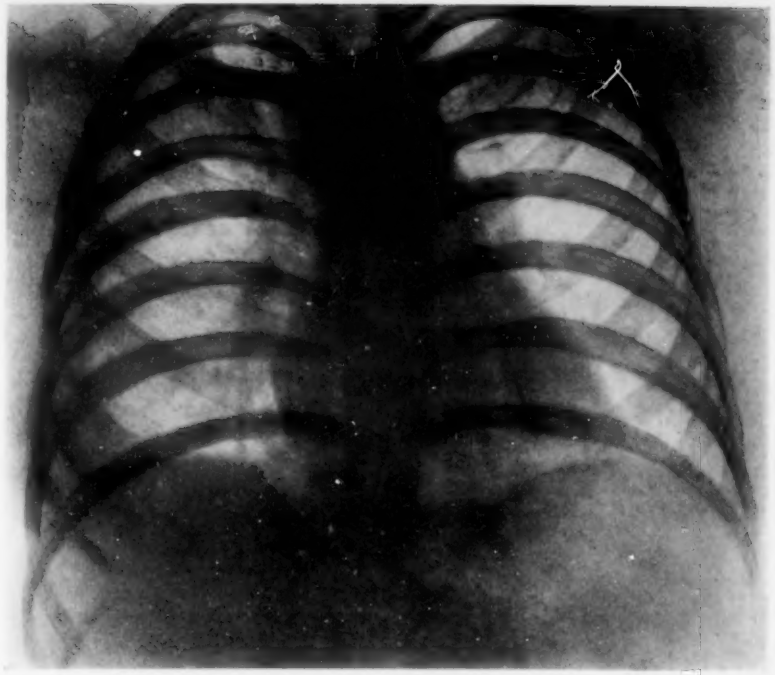

FIG. 4.—The end-result in the case shown in Fig. 3. (X-ray taken ten months after operation.) The cavity is obliterated. There is only a slight haziness to indicate what was found at operation to be a greatly thickened pleura.

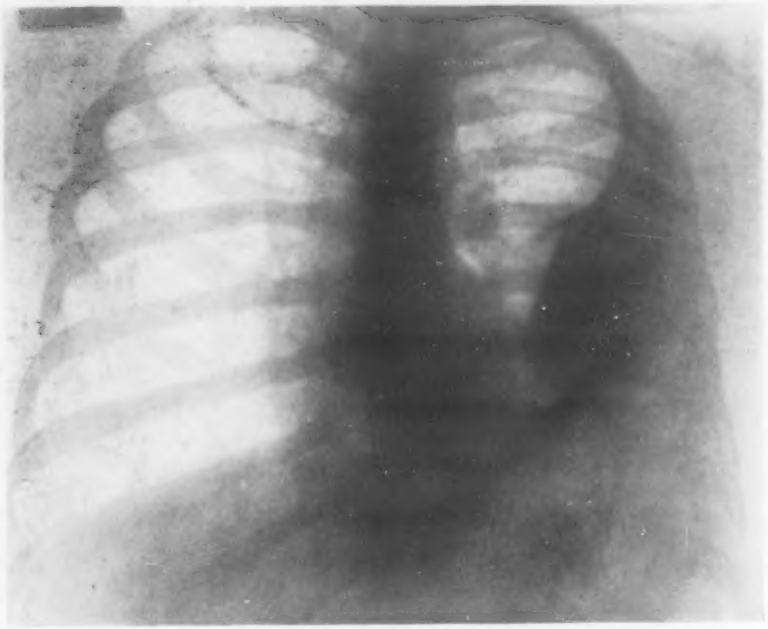

FIG. 5.—A chronic empyemic cavity treated simply by irrigations with Dakin's solution. (See text, Method 4.)

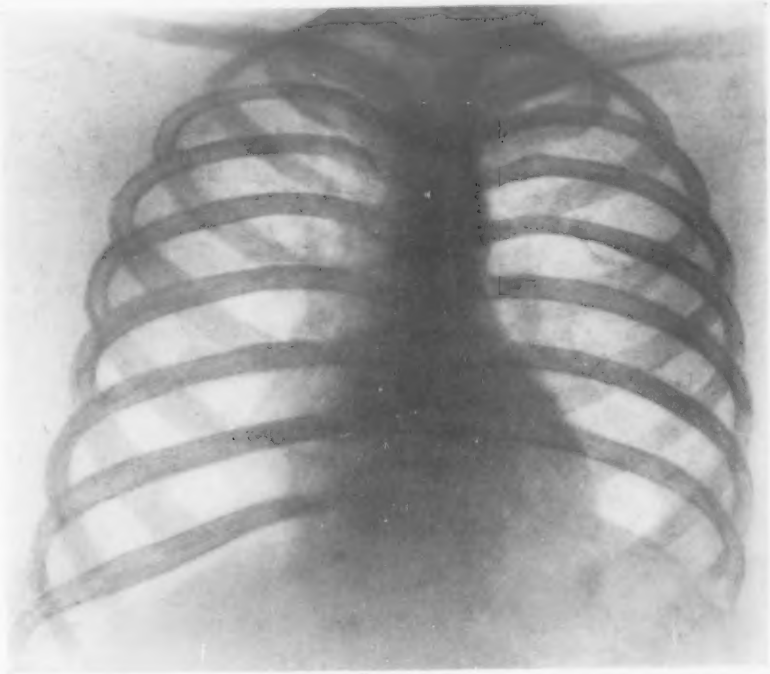

FIG. 6.—The end-result in the case shown in Fig. 5. (X-ray taken twelve months after beginning treatment.) The cavity is obliterated. The shadow cast by the thickened pleura has practically entirely disappeared.

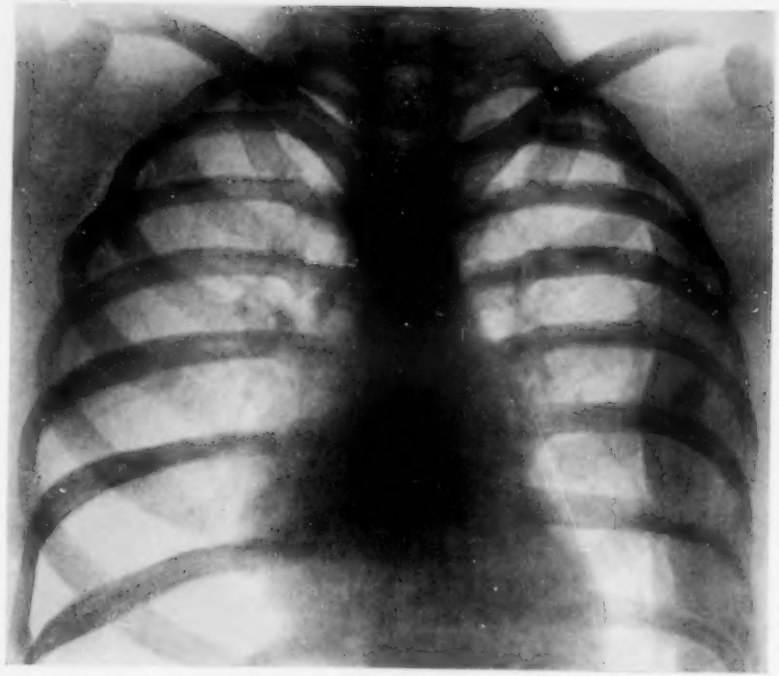

FIG. 7.—A chronic empyemic cavity treated by immediate sterilization and closure without drainage. (See text, Method 5.)

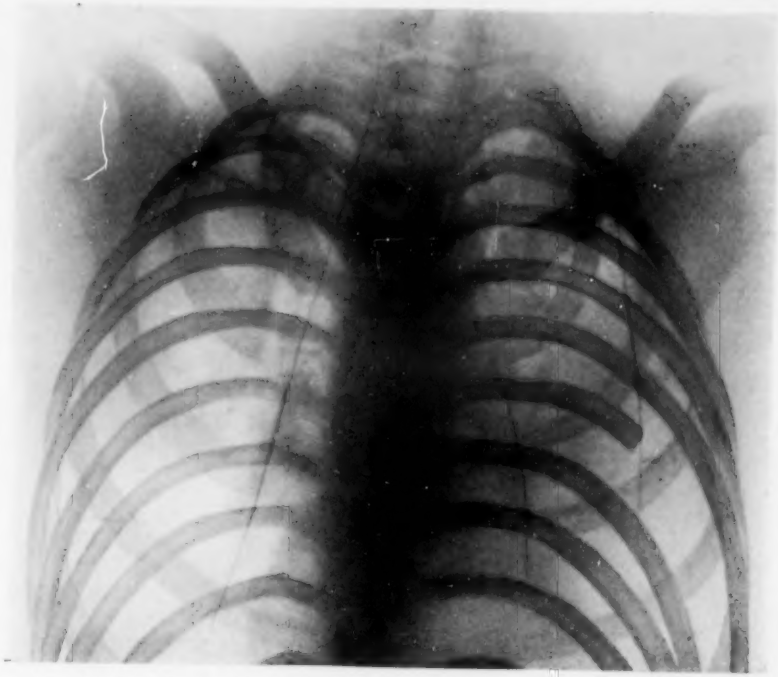

FIG. 8.—The end-result in the case shown in Fig. 7. (X-ray taken one month after operation.) The defect in the rib shows the extent of the rib resection in this case. The cavity is obliterated. The shadow cast by the thickened pleura has entirely disappeared.

especially in the long tubular intrapleural sinus tracts of tuberculous origin, we have been quite uniformly successful; and we have five cases at present which have been healed over long periods. Our total results, therefore, in seven cases are six successes and one failure. For the non-tuberculous cavities we have now, I believe, better methods of treatment; but if it is fair to judge from a small experience, the tuberculous cavities respond more quickly to bismuth paste than to Dakin's solution.

2. *Substitution of a Sterile Extrapleural Cavity for a Septic Intrapleural Cavity by Stripping the Parietal Pleura.*—It seemed possible from experiments on animals that an intrapleural cavity might be obliterated by stripping the parietal pleura according to the method of Tuffier and bringing it in contact with the visceral pleura. The contact of the two pleural surfaces might be maintained by suction upon the sinus or by the injection of air or nitrogen into the resulting sterile extrapleural cavity. A necessary requirement was that the sinus be at the lowermost point of the cavity. The operation was carried out away from the sinus tract and in a relatively aseptic field. After resection of 10 to 12 cm. of a single rib it was possible to strip the parietal pleura over a wide area. The procedure proved to be feasible if the parietal pleura were not too greatly thickened. In the presence of a rigid, board-like pleura, however, the result was that when mobilized the pleura stretched across the thoracic cavity as a cord subtends an arc, and could not be brought into contact with the visceral pleura. In two cases of this kind we were able to reduce the size of the cavities by one-half and three-fourths, but failed to completely obliterate them (Fig. 2). In one case the procedure was successful. In view of the probable failure of this method in many cases, it was abandoned. But our experience was not without value, for it taught us that the parietal pleura over chronic empyemic cavities may be stripped away from the thoracic wall, and that we had, therefore, a method of exposing the entire cavity through the resection of a single rib. It suggested also that the presence of the thickened parietal pleura probably largely prevented the expansion of the lung, and that the excision of this structure might, therefore, permit the expansion of the lung. It led to two of the following procedures (3 and 5).

3. *Excision of the Mobilized Parietal Pleura Over the Empyemic Cavity. Irrigation of the Extrapleural Cavity with Dakin's Solution. Secondary Closure.*—It appeared from our previous observations that the thickened pleura about an empyemic cavity probably prevented the obliteration of the cavity. By stripping the parietal pleura from the thoracic wall well beyond the limits of the empyemic cavity and excising it, it seemed to us that the cavity might more readily be obliterated. The result is an extrapleural cavity, presenting upon the mesial wall of which is an area of visceral pleura. The procedure, moreover, removes at least half of the infected pleural surface and allows the formation of granulation tissue from the thoracic wall. It is easier, I believe, than the Fowler or Delorme

operation. The Dakinization of the extrapleural cavity seemed necessary because of the presence of infection. In practice the operation has been carried out as follows: After stereoscopic X-rays of bismuth injections of the cavity have been made to determine the size and position of the cavity, the sinus tract is carbolized, encircled by an incision, and dissected down to the pleura. Ten to 12 cm. of a single rib are excised and the parietal pleura stripped away from the chest wall well beyond the limits of the cavity. With as careful an observance of aseptic technic as possible the parietal pleura is incised, the granulation tissue removed from the cavity, and its entire inner surface carbolized. The parietal pleura is then excised. The wound is closed except for an opening large enough to permit the two tubes of our Dakin irrigating apparatus. This procedure has been carried out in four cases; in two cases as a primary procedure, and in two cases as a secondary operation following the failure of other methods. In all the cases the method was successful. The cavities varied in size from 100 to 200 c.c. in volume, were elliptical rather than spherical in shape, were of long standing (one to four years), and were surrounded by a markedly thickened pleura. Sterilization of the cavities and closure of the sinus tracts were effected in one case in thirteen days, in one case in twenty-one days, in one case in forty days, and in one case in sixty-seven days. We have examined personally or have letters from all these cases. In none have the wounds reopened. The patients have now been well for from six months to one and a half years. We have a series of pre- and post-operative X-rays in three of the cases. Although we cannot state accurately how soon after sterilization the cavities were obliterated, we know that all were obliterated rather promptly—almost surely within a month. In the later X-rays of these cases the shadows indicating the thickened pleura have almost entirely disappeared (Figs. 3 and 4).

4. *Preliminary Correction of the Sinus Tract and Cavity if Necessary. Sterilization of the Cavity with Dakin's Solution. Secondary Suture or Spontaneous Closure of the Sinus Tract.*—In the cases in which bismuth X-rays showed a sinus leading directly into a single cavity, the tubes of our irrigating apparatus were introduced into the cavity without preliminary operative procedures, and the sterilization of the cavity attempted. In the cases in which a long or tortuous tract led into the cavity so that mechanical conditions for subsequent sterilization were unsatisfactory, a preliminary operation with excision of the sinus tract or the establishment of a new opening into the cavity at a point of election was performed. This method has been carried out in ten cases. In one case no preliminary operation was necessary. In one case the cavity was opened widely and the large open wound—the bottom of which was formed by the cavity—subsequently closed by secondary suture. In eight cases the sinus was excised and either enlarged or closed and a new sinus established. In two of these eight cases rib resections (in one case

segments of two ribs; in one case of four ribs) were performed. In these two cases—in which the cavities were under the scapula—the rib resections may have hastened the obliteration of the cavities; but could not have influenced greatly the sterilization of the cavities. The results in the ten cases are as follows: One case insisted on going home three weeks after irrigations of the cavity were begun. At the time of his discharge the bacterial count was very low, but the cavity was neither sterile nor closed. This patient has not been heard from. One case, the only instance in this group of tuberculous empyema (secondarily infected with a hæmolytic streptococcus), was discharged two and a half months after beginning Dakin's solution irrigations. The bacterial count was rapidly reduced from infinity to one to two organisms per microscopic field; but for a month we were unable to do better than this and feared to close the sinus because of the presence of a hæmolytic streptococcus. A letter from this patient states that he is at present in a tuberculosis sanitorium and that the cavity is still draining. In one case the cavity was bacteriologically sterile and closed in one month after beginning Dakin irrigations, but reopened two weeks later. A secondary operation with stripping and excision of the parietal pleura cured this patient. In one case sterilization and closure were accomplished in three weeks. The wound, however, reopened and discharged, then closed spontaneously, and has remained closed for one and a half years. In the remaining six cases sterilization and closure were accomplished in fourteen days, twenty days, twenty-three days, thirty days, sixty days, and seventy days. By personal examinations or letters we know that in none of these cases has the wound reopened and that they have been well for from seven months to three years. Exclusive of the three cases in which sterilization was not accomplished, we know from X-ray studies that the cavities were not obliterated at the time of closure of the sinus tracts; in other words, that we closed or allowed to close a sinus over an intrapleural cavity. We know from subsequent X-ray examinations of four patients who have since returned for examination that the cavities—even though surrounded by a markedly thickened pleura—have spontaneously been obliterated and that the thickening of the pleura has largely disappeared (Figs. 5 and 6). Our total results, therefore, in these ten cases have been six complete successes and four failures. In one of the four failures the sinus, which reopened two weeks after its primary closure, closed spontaneously and has since remained closed. In another case included as a failure in this group, a subsequent operation resulted in the closure of the sinus and a complete cure. In only two cases, therefore, did we fail in the closure of the cavities before the patients left the hospital.

5. *Immediate Sterilization of Chronic Empyemic Cavities with Pure Carbolic Acid. Closure without Drainage.*—Our experience with the progressive sterilization of empyemic cavities led us to attempt the immediate sterilization and complete closure of chronic empyemic cavities at

the time of operation. This procedure has been attempted only in small (up to 200 c.c. in volume) and favorably situated cavities. The operation has been carried out as follows: The sinus tract is carbolized, surrounded by an incision, tied off, and recarbolized. With a new set of instruments the sinus tract is followed down to the pleura, care being exercised not to open it. Ten to 12 cm. of a single rib are resected and the parietal pleura stripped from the thoracic wall beyond the limits of the cavity. With the wound held widely open with a rib spreader and with the greatest care not to contaminate the field, the parietal pleura is incised from one end of the cavity to the other (at the same time excising the sinus tract), and the cavity—previously cleansed by Dakin irrigations—opened widely. Holding up the edges of the incised parietal pleura the granulation tissue lining the cavity is wiped out and then the cavity is carbolized throughout with pure carbolic acid. The excess of acid is wiped out with alcohol or salt sponges. The parietal pleura has been treated in various ways; it has been allowed to simply fall into the cavity; it has been sutured to the visceral pleura, so as to bring the two layers of the pleura accurately into apposition; or it has been excised. The extrapleural cavity is wiped dry. The wound is closed without drainage. This procedure has been carried out in five cases, but in two cases operated upon before I went abroad, and successfully so far as immediate results are concerned, our records have not yet been found. In two of the three cases in which we have complete records the wound healed by primary intention and remained healed. In one case the wound healed per primum, but on the fifth day after operation an area of induration appeared in the subcutaneous tissues along the healed incision. Before the incision was opened cultures from the fluid in the cavity showed that the cavity was sterile. With the opening of the incision the cavity became secondarily infected, but was sterilized with Dakin's solution and later closed by secondary suture. By personal examination we know the end-results in these three cases. In the two cases in which the wounds healed by primary intention, the wounds have remained healed and the patients are well three and a half years and four years after operation. In the case which developed a secondary infection the wound has remained healed since the secondary closure of the cavity (Figs. 7 and 8). We know the fate of the cavities in these three cases. In one the cavity after operation became filled with blood, as proved by aspiration, but later was completely obliterated; in one, if we properly interpret the clear space in the X-rays, the cavity did not fill with fluid, but remained an air space until its obliteration (Figs. 9 and 10); in one the cavity remained filled with a blood-tinged serous fluid until its obliteration.

To summarize our observations, we may say that twenty-four patients with long-standing chronic empyema have been treated by one or another—in a few instances by two—of the methods described. There has been no operative mortality. Through the efforts of Miss Spicer, my

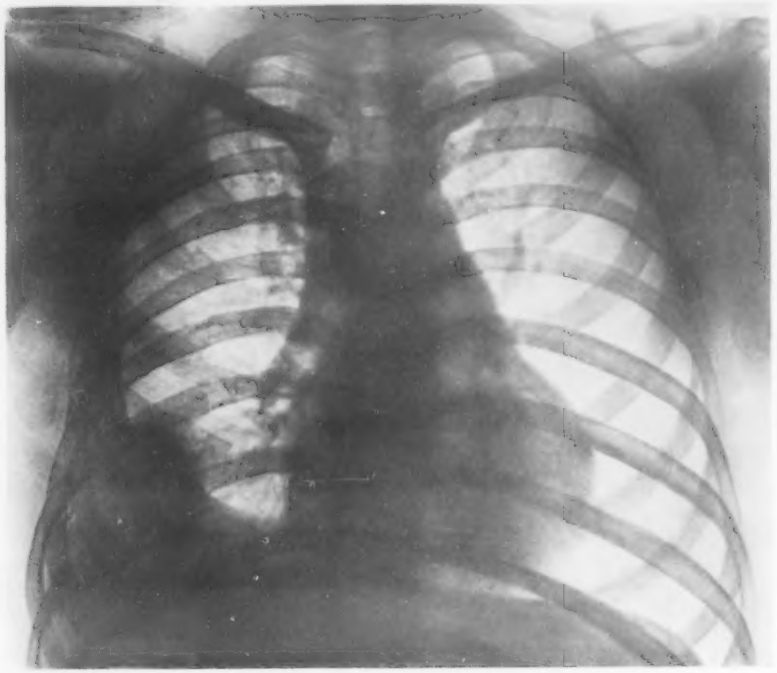

FIG. 9.—A chronic empyemic cavity treated by immediate sterilization and closure without drainage. (See text, Method 5.)

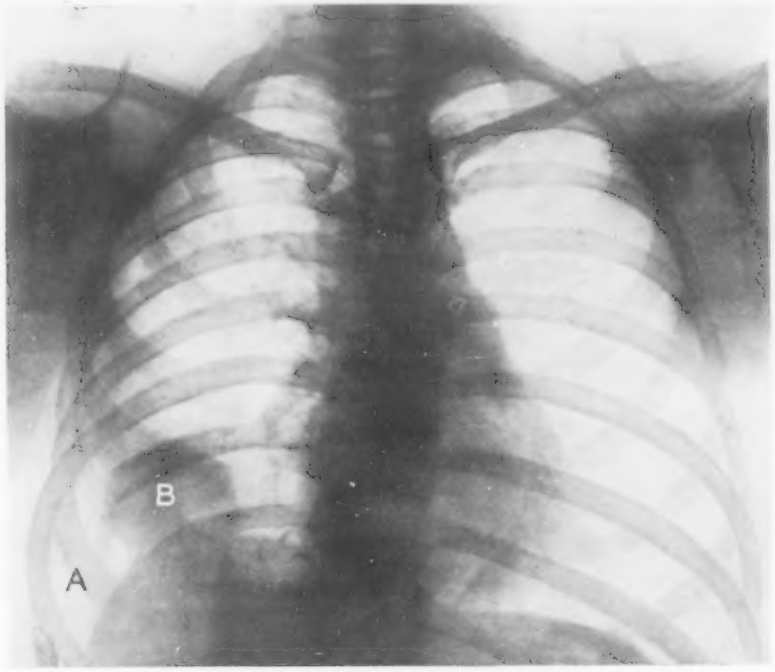

FIG. 10.—The result in the case shown in Fig. 9, three weeks after operation. The extrapleural cavity is not yet obliterated but is represented by the clear space, *A*. An area of thickened pleura is shown at *B*.

secretary, and Miss McEvoy, my social service aide, I have with one exception either examined personally or have received letters from all these patients. Twenty patients were discharged from the hospital with their wounds healed. In nineteen of these patients the wounds have remained healed, and eighteen patients are well from seven months to four years after their discharge from the hospital. One patient died of pulmonary tuberculosis two years after his discharge from the hospital; in one patient the sterile cavity became reinfected by the rupture into it of a tuberculous focus, and death occurred following an extensive thoracoplastic operation, three years after the sterilization of the cavity. Four patients were discharged from the hospital with their cavities infected and draining. Two of these patients are in tuberculosis sanitoria with their cavities still draining; one patient has since died of pulmonary tuberculosis. One patient discharged against our wishes three weeks after his admission has not yet been heard from. With the exception of this last patient, whom undoubtedly we could have cured had he remained in the hospital, our only failures occurred in patients with active pulmonary tuberculosis in whom a tuberculous empyema followed prolonged artificial pneumothorax treatment.

Not less interesting than the facts that empyema cavities of long standing can be sterilized and that subsequent to sterilization and closure of the sinus tracts they are spontaneously obliterated—and by expansion of the lung rather than by retraction of the thorax—is the fact that eventually there is almost a complete restoration to the normal. The late X-rays in our series show that the markedly thickened pleura certified at operation practically entirely disappears. In four cases we have indeed aided in this restoration to the normal by excising the thickened parietal pleura; but in cases in which such excision has not been done the thickened pleura has nevertheless disappeared (Figs. 3 to 9, inclusive).

In conclusion, we may add some observations on the complications following the irrigation of empyemic cavities with Dakin's solution. *Hemorrhage* from the cavity has occurred in three cases in the above series. It was not excessive nor alarming and spontaneously ceased so soon as the irrigations were discontinued. It invariably occurred late in the process of sterilization—at a time when the organism count was five per microscopic field or lower. It has not yet in our experience been a serious factor in the treatment of empyemic cavities by Dakin irrigations. In two cases the irrigations were discontinued for from twenty-four to forty-eight hours, then recommenced and without a repetition of the bleeding. In one case recurring, slight hemorrhages caused us to discontinue the irrigations and a single injection of bismuth paste was followed by closure of the sinus. *Bronchial fistulæ* have developed in the course of the irrigations with Dakin's solution in two cases. In one case there was a history of a previous bronchial fistula which had apparently been closed for three months. In the other case there was no history to

indicate that a bronchial fistula had previously been present. This complication has, in our experience, prevented for a time, at least, the continuation of the irrigations because of paroxysms of coughing, the taste of the solution, and the fear of strangulation. In the two cases in which bronchial fistulæ have occurred, injections of bismuth paste have been substituted for the irrigations.

EMPYEMA*

A SYLLABUS OF OPERATIVE TREATMENT

By Howard Lilienthal, M.D.
of New York, N.Y.

This is an attempt to standardize the selection of operative methods in the surgery of empyema of the thorax. It is presented here for discussion and criticism. It consists of: (1) A classification of the conditions demanding operation. (2) A list of procedures with a brief description of each. (3) A synoptic table showing the types of the disease paired with their appropriate operations.

In such a presentation as this there can be given no complete plan for carrying any save the simplest cases to their conclusions; but complications, such, for example, as the discovery of an unsuspected sacculation, may put the case in another class which can be treated according to the table. It is also recognized that there can be no absolute rule for the treatment of any given case because the conditions are never twice exactly alike; but this does not forbid our attempt to construct a model. Bacteriology is not considered because it has nothing to do with our subject. It is of prognostic value only. Every case is assumed to have been röntgenologically studied except in acute intrathoracic tension, when therapeutic aspiration is urgent even without the X-ray. Anæsthesia in minor operations should be local, even with rib resection. Intrapharyngeal differential pressure by ether vapor or nitrous oxide and oxygen is preferable for the more serious procedures. The Carrel-Dakin method is recommended as a most valuable post-operative aid but it should not be employed when the instillation of even small quantities of the fluid with a wide opening for its escape from the thorax is followed by severe coughing. Also in children and in a few susceptible adults the long-continued application of this treatment seems to cause a general deterioration. Such patients improve on discontinuing this remedy. Empyema can best be treated in a hospital.

Classification of Conditions Demanding Operation

(Tuberculosis, syphilis, actinomycosis, etc., are not included.)

Acute empyema	*A.* Seropurulent effusion	*a.* General or large. *b.* Sacculated, single or multiple. *c.* With purulent expectoration (hidden lung abscess or empyema emptying through a bronchus).
	B. Frankly purulent effusion	*d.* With lung abscess (intrinsic). *e.* With lung abscess (bronchiectatic). *f.* From extrapleural sources other than lung, by direct extension. *g.* With tension pneumothorax. *h.* Traumatic.

* Read before American Surgical Association, May 3, 1920.

Chronic empyema	I. With closed thorax. II. With pleurobronchial fistula and closed thorax. III. With open thorax (fistula). IV. With pleurobronchial fistula and open thorax. V. With fibrosis and permanent contraction of lung.

A List of Procedures with a Brief Description of Each

Procedure No. 1.—Diagnostic Aspiration. Only a few c.c. to be removed. Syringe to be detached and needle to be withdrawn while 2% lysol is slowly injected through it into the puncture tract to prevent infection and phlegmon of the chest wall (5 to 15 c.c. may be injected).

Procedure No. 2.[1]—Therapeutic Aspiration. Use fine trocar and canula with rubber tube attached so as to empty the chest by patient's expulsive efforts and by gravity. No forcible suction to be employed. Air permitted to replace the fluid removed or if desired the air may be expelled from the chest by the patient's straining with closed glottis or even by his normal respirations, the tube being pinched during *inspiration* until no more bubbles appear on straining or on *expiration* from end of tube held under water. The canula is then quickly withdrawn. (X-ray will demonstrate the efficacy of this method of getting air out of the thorax.)

Procedure No. 3.—Minor intercostal thoracotomy and tube drainage (with or without airtight closure).

Procedure No. 4.[2]—Resection of rib with its periosteum. Tube drainage.

Procedure No. 5.[3]—Major intercostal thoracotomy with rib retraction (rib spreader) and full exploration of chest cavity with mobilization of lung if desirable.

Procedure No. 6.[4]—Noncollapsing major thoracoplasty, with costotomy but no resection of ribs. Mobilization of lung.

Procedure No. 7.—Various forms of collapsing thoracoplasty. (Schede, Estlander, Wilms, etc.)

Synoptic Table

(Showing the Types of the Disease Paired with Their Appropriate Operations.)

Condition	Operation
Acute Seropurulent Effusion.	
a. General or large	Procedure No. 2. Repeat if necessary until frank pus is present or no more fluid accumulates.
b. Sacculated, single or multiple.....	Procedure No. 2 applied to larger cavities. Repeat if necessary until pus is present or no more fluid accumulates.
c. With purulent expectoration (hidden lung abscess or empyema emptying through a bronchus)..	Procedure No. 2. Repeat until pus is present (or no more fluid accumulates), then Procedure No. 5.
d. With lung abscess (intrinsic)....	Procedure No. 4. This is preliminary as a rule, but it may prove curative.

OPERATIVE TREATMENT OF EMPYEMA

Condition	Operation
e. With lung abscess (bronchiectatic)	Procedure No. 5 with a view to dealing later on or at the same time with pulmonary condition (lobectomy).
f. From extrapleural sources other than lung by direct extension...	Procedure No. 4. Generous resection and dealing at once with the cause (*e.g.*, subphrenic abscess).
g. With tension pneumothorax.....	Procedure No. 2 followed by Procedure No. 3.
h. Traumatic	Procedure No. 2 followed by Procedure No. 3 or 5 according to extent of trauma.

Acute Frankly Purulent Exudate.

a. General or large	Procedure No. 3 followed if course is unsatisfactory by Procedure No. 5. (Fluoroscopic study important.)
b. Sacculated, single or multiple	If single, Procedure No. 4. If multiple, Procdure No. 5.
c. With purulent expectoration (hidden lung abscess or empyema emptying through a bronchus)...	Procedure No. 4.
d. With lung abscess (intrinsic)	Procedure No. 4. Generous resection with simultaneous or deferred drainage of abscess.
e. With lung abscess (bronchiectatic)	Procedure No. 5. Possibly as first stage of lobectomy.
f. From extrapleural sources other than the lung by direct extension.	Procedure No. 4 with immediate attention to cause.
g. With tension pneumothorax.....	Procedure No. 2, later No. 3 or No. 5, according to X-ray.
h. Traumatic	Procedure No. 4. (Revision probably necessary.)

Chronic Empyema

I. With closed thorax (simple).....	Procedure No. 5.
II. With pleurobronchial fistula and closed thorax. (X-ray diagnosis pyopneumothorax.)	Procedure No. 3, later No. 5.

Condition	Operation
III With open thorax (fistula)	Procedure No. 6.
IV. With pleurobronchial fistula and open thorax (thoracic fistula)	Procedure No. 6.
V. With fibrosis and permanent contraction of lung. (Demonstrated at operation by impossibility of inflating lung with intrapharyngeal pressure after "decortication"; and after weeks of further effort by blowing and coughing exercises)	Procedure No. 7.

POSTSCRIPT.—The use of Beck's paste or of 5 to 10 per cent. iodoform in vaseline is recommended in certain narrow cavities which have previously been rendered bacteria free or nearly so. This form of treatment is often successful even when there is an open bronchus at the end of the tract.

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FATAL POST-OPERATIVE PULMONARY THROMBOSIS*

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It has seemed worth while to consider our experience with post-operative pulmonary thrombosis in connection with a review of the most important papers which have been written upon this subject with a view to bring out any points of value in the direction of prophylaxis, as none of the methods of treatment of the condition, once it has been established, seem to have proved successful.

It seems clear from all of the many careful observations that are recorded in the literature, as well as from our own experience, that the condition does not depend upon any one cause, although most observers seem to agree upon several causes which are more important than others. In order that these views may be brought out clearly, we have stated each together with the name of its author in as concise a form as possible before stating our own observations and conclusions.

Causation.—The causes of pulmonary thrombosis in their order of importance are: (1) Local infection; (2) anæmia; (3) slowing of blood stream; (4) subnormal general physical condition; (5) cachexia; (6) micro-organisms in the blood; (7) excess of white blood-cells; (88 inefficient hæmostasis; (9) traumatization of tissues with retractors, etc.; (10) injury to veins of extremities due to badly arranged operating table; (11) injury to intima of veins; (12) excess of calcium salts in the blood.

History.—The condition has been recognized and discussed for generations. Van Swieten [8] in 1705 recognized that clots occur in the vessels during the puerperium and wrote gravely on their prognosis.

In 1784 Dr. Charles White, a distinguished London physician, did not associate pulmonary embolism with phlegmasia alba dolens.

Virchow in Berlin and Meigs in Philadelphia wrote on the subject of blood clots stopping the stream of the circulation; Virchow advocating that the obstructing clot must always travel to the heart and the pulmonary arteries; Meigs advocating that it may be formed *in situ* in the heart or pulmonary artery as well. Virchow upheld the embolus theory, Meigs the thrombosis theory.

Virchow [29] first showed the relation between thrombi and emboli, pointing out that emboli not infrequently have their origin in the soft-

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ening, breaking down, and detachment of venous thrombi. He stated that in embolism the plug consists, not of blood clots, but of so-called vegetation or concretion of fibrin which has been washed off from the valves of the heart or from the endocardium and carried forward by the arterial current until the vessels become too small in calibre to allow it to advance any further.

Anatomy.—Cohnheim and Litten [9] experimentally demonstrated the fact that the arterial ramifications of the pulmonary artery do not anastomose. They are end-arteries.

The bronchial arteries supply the parenchyma of the lung with not insignificant quantities of blood. Virchow called them the nutrient arteries of the lung. They showed that pulmonary infarcts do not result in the death of the lung, when death of the animal itself does not result, but prevent the functioning of the infarcted area for purposes of oxidation. They conclude that despite the absence of parenchymatous changes in infarcted lungs, where the quantity of infarcted lung is sufficient, death must result from disturbance of respiratory function. The bigger the obstructed artery or the larger the number of smaller obstructed arteries, the greater is the effect upon respiration, and so it happens that where multiple emboli occur successively, even without parenchymatous changes in the lungs, death must eventually ensue from respiratory insufficiency.

Anningson [1] claimed that in the pulmonary artery the clots are most commonly found at the point where it breaks up into its branches.

Mann [10] states that deaths due to pulmonary embolism should be divided into three groups: (1) Immediate death occurring when only a small portion of the pulmonary circulation is obstructed. (2) Death caused within a few minutes and due to complete or almost complete blocking of the pulmonary circulation. (3) Delayed death, the result of an increase, by thrombosis, of an initial blockage by an embolism of a portion of the pulmonary circulation.

Experimentally, it was impossible to produce death or seriously imperil the life of the animal by emboli until the pulmonary circulation was greatly obstructed.

There is a wide difference of opinion regarding the frequency with which pulmonary thrombosis occurs primarily or is due to an embolus.

Virchow [20] divided obstruction of the pulmonary artery into four groups:

1. Following compression of a branch of pulmonary artery, coagulation of the blood may occur.

2. The introduction of a deleterious substance sets up an inflammation and this causes coagulation of the blood.

3. Spontaneous coagulation of blood from causes within the blood itself.

4. Obstruction occurs through a more or less compact mass which is carried into the pulmonary vessels and becomes lodged in them.

The majority of plugs in the trunk or main divisions of the pulmonary artery found in cases of sudden death, present the anatomical characters of emboli, associated perhaps with secondary thrombi, but there remain a certain number of cases of sudden or gradual death from primary thrombosis of the pulmonary artery or from thrombosis extending into a main division from an embolus in a smaller branch.

Welch [21] showed that bland embolism in medium-sized and small branches of the

pulmonary artery in normal lungs and without serious impairment of the pulmonary circulation usually causes no symptoms and no changes in the parenchyma of the lungs.

Virchow [20] pointed out that emboli fill the pulmonary arteries without adhering to the walls when they are young, while the vessel walls show no changes, but when the emboli are old they cling to the wall which shows inflammatory changes.

Emboli usually stop at the bifurcation of a large artery, usually the vessels of the second and third order (Paget). Before and behind these fresh blood coagulation may occur.

Welch[22] shows that an embolus is the starting-point of a secondary thrombosis, which usually, although not always, completes the closure of the vessel, if this was not affected by the embolus itself, and extends on each side to the nearest branch.

Humphrey [3] said thrombosis occurred most commonly at the junction of the profunda and saphena veins with the femoral. The presence of valves at the meeting-point of two veins helps to bring about the condition.

Anningson [1] thinks that inflammation might follow the clotting, but was not the cause of it.

Clots may be absorbed, leaving the veins as normal as before.

Glynn [24] believes that emboli in the pulmonary artery may be distinguished from primary thrombi by the fact that the former are coiled or fractured or riding astride of a bifurcating vessel.

Fowler and Godley hold that embolism is commoner than thrombosis (pulmonary), but spontaneous thrombus does occur. West considers spontaneous thrombosis exceedingly rare.

Welch [22] believes in the usually accepted opinion that the majority of plugs found in the pulmonary artery and its main divisions in cases of sudden death are emboli.

Recently, Mery [14] in France and Newton Pitt in this country have maintained that spontaneous thrombosis is not infrequent, that in fact it may be commoner than embolism.

Box,[5] after making careful autopsies of several cases of so-called pulmonary embolism, came to the conclusion that these cases are a combination of thrombosis with embolism. He considers that a clot first forms in the pulmonary artery or in the right side of the heart and some sudden movement causes detachment of this clot which enters and completely plugs one or both pulmonary arteries.

He explains that a clot forming in the pulmonary artery or right ventricle very soon after the operation may become absorbed without producing symptoms, but it may increase in size, become detached and then plug the pulmonary artery.

Wilson [28] thinks it is probable that obstruction of the pulmonary artery sometimes occurs without proving fatal.

According to Foulkrod,[13] embolism of the pulmonary artery is invariably fatal. When involving the small blood-vessels in the lungs it may produce atalectasis, infarct, abscess or gangrene and the patient may recover.

Welch[22] states that asphyxia, cerebral anæmia, or interference with the coronary circulation are the factors concerned, but the exact apportionment to each of its due share in the result is not easy, nor very important.

Dr. Wm. Zahn [35] made a very extensive study of the formation of thrombi. His work will always stand out as a classic upon this subject and should be studied in detail in considering this subject.

Incidence.—In McLean's [29] experience emboli and embolic abscesses follow 2.2 per cent. of all laparotomies.

Ritzman [34] reported fifty-five cases in 6000 autopsies. All patients were over thirty years of age, and most were between fifty and seventy. There were as many females as males.

Bang[8] reported eighty-eight cases of embolism, one-half of which occurred in persons between twenty and thirty years of age.

In Lichtenberg's[22] collection of 23,680 operations, including 16,000 laparotomies, there were 2 per cent. of pulmonary complications and among the laparotomies 5.5 per cent.

Among 1000 operations on the vermiform appendix, Muhsom[23] reported thirty-one cases, five of which were fatal.

Mauclair[23] has collected fifty cases after the radical cure of inguinal hernia, twelve out of twenty-five fully reported being fatal.

The prognosis of post-operative pulmonary embolism is extremely grave; in 233 cases collected by Lenormant, death occurred in 106 or 45.5 per cent.

The order of frequency in which emboli are found in the different arteries may be given as follows: pulmonary, renal, splenic, cerebral, iliac and lower extremities, axillary and upper extremities, cœliac axis with its hepatic and gastric branches, central artery of the retina, superior mesenterics, inferior mesenterics, abdominal aorta, coronary, and the heart.

Lotheisen[13] collected sixty-six cases of pulmonary embolism, of which fifty-five were fatal:

1. Following fractures, thirty-six cases; thirty fatal.
2. Contusion, six cases; five fatal.
3. Tendon and muscle lacerations, four cases; four fatal.
4. Following operations, twenty cases; sixteen fatal.

Of Lotheisen's sixty-six cases, only six were under thirty, the majority being over forty. Of his sixty-six cases, forty-one were males, twenty-two females, and three unspecified.

Bang[8] found ten cases of embolus in 600 autopsies—1.66 per cent.

According to Wilson,[25] in operations on blood-vessels, alimentary canal, and genito-urinary organs, from 1 to 2 per cent. of all cases give more or less distinct evidence of emboli, about 7 per cent. of which are in the lungs.

About 10 per cent. of post-operative emboli which give clinical symptoms cause death.

Foulkrod[13] found thirty-seven cases of proved or suspected pulmonary embolism. According to his observations, thrombosis and embolism (not confined to pulmonary) occur in from 1 to 3 per cent. of cases, including both post-operative and obstetric. Obstruction of the pulmonary circulation alone occurs in a very much smaller percentage of cases.

Beckman, quoted by Van Sweringen,[23] reports six cases of pulmonary embolism occurring at Rochester in the first eight months of 1910 out of 4530 consecutive cases operated upon.

Kelly and Cullen report four deaths from pulmonary embolism out of 901 hysterectomies.

Howard showed that venous thrombosis occurred thirty-four times in 3774 patients with appendicitis and that a little less than one-eighth of these were cases of pulmonary embolism.

Albanus found that pulmonary embolism followed in 2 per cent. of abdominal operations.

Burkhard gives in 236 operations for uterine fibroids twelve cases of embolism (Keen's Surgery).

In Bidwell's[8] practice pulmonary embolism or thrombosis has occurred in .5 per cent. of abdominal operations.

The risk of pulmonary thrombosis after an appendectomy in the quiet stage is not merely a nominal one, since three such cases occurred in Bidwell's practice and represent a mortality of nearly 1 per cent. after operation, the death rate from other causes being only .4 per cent.

Out of 700 obstetrical cases, Church[9] saw only one case of pulmonary embolism. These cases are evenly divided between primiparæ and multiparæ.

Playfair[23] states that as far as present statistics go, thrombosis and embolism seem more common in primiparæ than multiparæ.

Lotheisen[15] states that emboli occur, as a rule, in adults. Only one case of a child with pulmonary embolism could be found in the literature—a year-and-a-half-old boy with diphtheria and pneumonia.

Bidwell[6] thinks that pulmonary embolism after abdominal operations may be more frequent than is generally supposed and that its apparent rarity may be due to the surgeon's natural dislike to attract attention to his fatal cases.

McLean's[20] studies and observations have convinced him that a far greater percentage of post-operative ailments than we are accustomed to attribute to embolism really owe their origin to that cause.

Etiology of Thrombosis.—Welch[28] states that slowing and other irregularities of circulation in combination with lesions of cardiac or vascular wall or with the presence of microörganisms or other changes in the blood are important predisposing causes of thrombosis and frequently determine the localization of the thrombus.

2. Changes which impair or destroy the smooth surface of the normal inner lining of the vessel play an important part in the etiology of thrombosis (inflammation, atheroma, calcification, necrosis, other degenerations, tumors, compression and injury).

3. Infective thrombi (thrombophlebitis) develops during the progress of pneumonia, typhus, acute rheumatism, erysipelas, cholera, scarlatina, variola, tuberculosis, syphilis, nearly all acute and chronic infections. Likewise in chlorosis, gout, leukæmia, senile debility and chronic wasting, and cachectic diseases, particularly cancer, thrombosis is a recognized complication.

4. Chemical changes in the blood-ferment thrombi. Alterations in the formed elements of the blood caused directly or indirectly by toxic substances are of great significance in the etiology of pulmonary thrombosis.

a. Increase of blood platelets—there is a parallelism between the disposition to thrombosis and the number of platelets in certain diseases.

b. Calcium content is an important factor in coagulation of blood.

As factors in the causation of thrombosis, Lotheisen[15] considers:

1. A change in the blood constituents.
(*a*) Anæmia (puerperal hemorrhage or uterine hemorrhage due to myoma uteri).
(*b*) Gestation (physiological changes in blood and leucocytosis).
(*c*) Chlorosis.
(*d*) Prolonged fevers, malaria, cholera.
(*e*) Elephantiasis (Fayrer, three cases).
(*f*) Cachexia-malignant tumors.
2. Slowing of blood stream.
(*a*) Weak heart.
(*b*) Fatty degeneration—pregnancy.
(*c*) Pregnancy—gravid uterus pressing on veins.
3. Change in the vessel wall.
(*a*) Phlebitis.
(*b*) Trauma.
(*c*) Atheroma.

Wilson[32] states that the most important factors concerned in extensive post-operative thrombosis are:

(*a*) Injury of vascular walls.
(*b*) Slowing and stagnation of blood-stream.
(*c*) Disintegration of the corpuscles of the blood from toxic substances.
(*d*) Bacteræmia.

Foulkrod[13] enumerates as pathological conditions influencing coagulation the fol-

lowing: Preëxisting thrombosis, toxæmia or infections—under this heading he includes infections of the endometrium and broad ligament veins and particularly bronchial infections, profound mental depression, placenta prævia or other conditions producing excessive hemorrhage, mechanical pressure from the weight of the uterus, and slowing of the heart action.

Playfair [29] believes further thromboses of heart and pulmonary artery are sometimes due to dysentery; typhus and typhoid fever may also cause death by thrombosis of pulmonary artery.

According to Ritzman's [34] observations, the causes were various: vitium cordis, myodegeneration, arteriosclerosis, anæmia and cachexia, conditions causing phlebitis, injuries and operations. Nearly all the affections were related to vascular disease of some sort.

Church [8] claims that thrombosis is induced in puerperal state, rheumatism, fevers and other blood dyscrasiæ, such as erysipelas, diphtheria, pneumonia—increase of fibrin in the blood one third.

Bidwell [6] says pulmonary embolism is unpreventable since the active cause as well as the means of prevention are not known.

It is the opinion of Bidwell that thrombosis occurs only in consequence of changes in the blood-vessels, the blood, or both. A thrombus, of course, is formed by the development of fibrin; but fibrin does not exist in healthy blood, but is produced by the action of fibrin ferment on fibrinogen. Fibrin ferment does not exist in the blood, but is the result of a combination of calcium salts with nucleo-proteid. Calcium salts are normal constituents of the blood, but nucleo-proteid is not, and it is probably produced by degeneration of leucocytes and of blood platelets. In normal circumstances a considerable quantity of nucleo-proteid can be disposed of in the circulation, probably by the action of the endothelial lining of the blood-vessels; this power is, however, diminished by injury, by inflammation, and by retarding of the blood-stream.

Therefore, thrombosis forms when the walls of the blood-vessels have been injured; also in cases of sepsis, by increase of CO_2 in the blood, by general conditions, such as chlorosis and anæmia, and lastly by specific fevers, more especially typhoid.

Bland Sutton,[6] in his Hunterian lecture, is convinced that the formation of thrombi in the great veins after pelvic operations is due in all cases to sepsis.

Faure [11] describes two fatal cases occurring within a few days operated between February 12 to 24, 1919, during the influenza epidemic after the clinic had been free from such fatalities for many months, thus attributing these cases to influenza infection. At the same time, there were three additional cases of phlebitis and numerous cases of wound infection during this period, while the clinic had been free from all of these complications for months.

McLean [20] has shown how, except in the presence of an infection, it was impossible to produce experimentally a thrombus and a resulting embolus, and for this reason claims that a perfectly aseptic operation is rarely followed by an embolic process.

He insists that endothelial damage, on which so much stress is generally laid, is not, *per se*, a cause of thrombosis. Infection and necrosis (or the toxins derived from an infection and necrotic process) are probably the most important factors in thrombus production. A slowing of the blood-stream is a contributory cause, but of itself will not cause a thrombus to form.

Fromme [12] introduced a silk thread into the jugular vein of rabbits. A sterile thread produced a thrombosis only in anæmic animals or in those in bad physical condition, while the thread impregnated with any form of bacteria regularly produced thrombi.

Talke [36] said he had placed culture of staphylococci near thirteen arteries and thirty-one veins in thirteen animals, and he removed these after nine to twelve hours. Twenty-two veins and eleven arteries were thrombosed. The vessel wall and the surrounding tissues showed typical inflammation, but the thrombosis occurred before

the microörganisms had entered the lumen of the vessel, hence seemed to be caused by the toxins.

Bidwell [5] quotes some surgeons who assert that all cases of thrombosis are really septic in origin, but states that it is difficult to agree with this statement, since it occurs in aseptic cases and in such a case as a gastric ulcer.

Libanoff [17] explained the formation of thrombokinase in large hæmatomata leading to the danger of local or distant thrombosis and pointed out the necessity of evacuating these to avoid their absorption. Commenting on this, Murphy asks: "Why has one clinic so many cases of thrombosis and embolism and the other clinic so few?" May it not be due to imperfect hæmostasis by one operator and the perfect capillary hæmostasis of the other? In the first instance, large hæmatomata form, and the latter give up the thrombokinase and thrombosis follows; while in the other no hæmatomata form and consequently no thrombokinase is given out, and therefore no activity of thrombogen occurs with the calcium salts to form a thrombus.

Volker [20] said he was surprised to find in necropsies on animals dead of embolism that there was invariably some brownish liquid blood in small quantities at the bottom of the wound, and it occurred to him that there might be some relation between the debris of these small hæmatomata and fatal embolism. He believed the debris from the hæmatomata should be looked upon as the cause of embolism.

Dr. Joseph Price, quoted by Volker,[20] called attention to the dangers of these small "blood pools" in the pelvis following operations as early as 1893 and placed great stress upon the danger from them and the importance of leaving the field of operation perfectly dry.

After delivery the blood dyscrasia is increased by the absorption of effete matter in the process of uterine involution. Severe hemorrhage and syncope with slowing of the blood-stream increase the tendency to coagulation. Plugs normally found in natural labor in the open orifices of the uterine sinuses after separation of the placenta may find their way into the systemic circulation.

Zurheile [20] claims that retardation of blood-stream is the main factor in the production of a thrombosis, so that the blood plates pile up mechanically in the more sluggish blood. His experiments show the uselessness of striving to prevent thrombosis by reducing the coagulating property of the blood; as we are unable to act on the blood plates, all we can do is to prevent the blood-stream from becoming sluggish.

Aschoff [3] suggests that it may be possible to prevent thrombosis by changing the physical condition in the circulation, combating any tendency to slower pulse rate. He does not think that thrombosis is always of infectious origin, but a superimposed infection transforms a primary insignificant thrombosis into a dangerous thrombophlebitis. He supports the theory advanced by Virchow that the slowing up of the stream is the principal factor in the development of thrombi.

All cases observed by Glynn [14] of pulmonary thrombosis had been bed-ridden, and passive congestion of the lungs may have been a predisposing cause of thrombosis. It is also a fact that nine cases had been anæsthetized.

Bardeleben [4] showed that streptococci introduced into the blood-stream produce a thrombosis only if the stream is slowed down, otherwise non-virulent varieties are destroyed, while virulent varieties produce severe bacteræmia if highly virulent streptococci become lodged upon the vessel wall.

Bidwell [5] points out the fact that when thrombosis attacks a femoral vein, the left is most usually affected, and this is explained by the course of the left iliac vein being less direct than that of the right and also by the fact that the flow of blood through it is likely to be retarded by the pressure of a loaded sigmoid.

Playfair [20] believes that central thrombosis (cardiac and pulmonary) should be looked upon as a complication which is liable to attend the performance of surgical operations in general, but more especially those done on cachectic subjects or those involving much shock or hemorrhage.

He also states that both thrombosis and embolism are much more common in patients who are anæmic and weak either from hemorrhage or other cause.

Duncan[8] argues that it is due to anæmia, which so frequently affects women suffering from uterine fibroids, and supports his views by quoting a case of a woman with fibroids dying from pulmonary embolism while in the hospital awaiting operation.

Clark, quoted by Van Sweringen,[25] believes that thrombosis in non-septic epigastric veins is due to propagating thrombus of the deep epigastric veins originally produced by the traumatism resulting from operative manipulations and especially the use of heavy retractors.

Others maintain that post-operative thrombosis is caused by the pressure of retractors on the edges of the abdominal wound causing injury to the deep epigastric veins; the thrombus forms first at the seat of injury and afterwards spreads down to or around to the femoral vein.

It is generally supposed that patients suffering from uterine fibroids are peculiarly liable to thrombosis and embolism; this is explained (1) by the increase of calcium salts in the blood, as shown by the tendency to calcareous degeneration of the fibromata, and (2) by some degeneration and weakening of the cardiac muscle fibre which is commonly associated with the condition and, according to several authors, to the fact that the stump left after the removal of the uterus has not been carefully covered with peritoneum after accomplishing perfect hæmostasis, thus favoring infection.

Anningson[1] thought it possible that the tendency to coagulation during the puerperal state and in pneumonia, erysipelas, etc., was due to excess of white corpuscles in these conditions.

Virchow[29] believed that the fibrin clots or pulmonary emboli were secondary to thrombosis elsewhere. This thrombosis occurs in the veins or right heart, and is carried to the pulmonary arteries by the blood-stream.

To substantiate this he reports that out of seventy-six sections performed in August in the Morgue of the Berlin Charities he encountered eighteen venous thromboses and six lung or pulmonary thromboses.

Obstruction may or may not cause change in the parenchyma.

When the blood in one vein clots, the coagulum extends beyond the mouth of the next vein, so that, as the blood from this vein passes by, small pieces of coagulum can easily be detached. Virchow has been able to match the edges of pulmonary emboli with those of venous thrombosis from which they originated.

Wilson[28] states 80 per cent. of emboli have their source in venous thrombosis, 10 per cent. are cardiac and 10 per cent. scattered and undeterminable. Long, loosely formed thrombi from medium-sized veins are those chiefly concerned in embolism. When large, loose thrombi are formed in resting patients, any unusual exertion or change of position may cause a dislocation of large masses which become dangerous emboli.

Church,[8] quoting Playfair in his "Science and Practice of Midwifery," says, "I have shown from a careful analysis of twenty-five cases of sudden death after delivery, in which accurate post-mortem examination has been made, that the cases of spontaneous thrombosis and embolism depend upon the period after delivery at which the fatal result occurs. In seven of these cases there was distinct evidence of embolism, and in them death occurred at a remote period after delivery, in none before the nineteenth day. This contrasts remarkably with the cases in which post-mortem examinations afforded no evidence of embolism. These amounted to fifteen out of

twenty-five; in all of them, with one exception, death occurred before the fourteenth day, often on the second or third. The reason for this seems to be that in the former, time is required to admit of degenerative changes taking place in the deposited fibrin, leading to separation of an embolus; while in the latter, the thrombosis corresponds in time and to a great extent no doubt also in cause to the original thrombosis from which in the former the embolus was derived. Many cases I have since collected illustrate the same rule in a very curious and instructive way."

Playfair [22] expresses the following opinion:

Obstruction of the pulmonary artery after delivery may depend upon either embolism or spontaneous thrombosis.

The former usually occurs at a much later period after delivery than the latter, and spontaneous thrombosis probably corresponds with and is due to some cause similar in its nature to that which produces the obstruction of the peripheral veins in true cases of embolism.

Church [8] states thrombosis may occur simultaneously at the periphery (veins) and centre (heart and lungs) of blood-stream. Dr. W. J. Playfair cites cases of pulmonary obstruction that have not proved fatal immediately and in which shortly afterwards phlegmosia dolens commenced, showing thereby that coagulum was first formed in the centre and then at the periphery.

Thrombosis rarely occurs till one week after an operation; from the tenth to the fourteenth day is the usual time, but sometimes it is as late as one month. Recovery is generally complete in from two to three weeks. As a rule, these cases do not suffer from pulmonary complications, and in those rare cases in which sudden death does occur after a femoral thrombosis, it is probable that a thrombosis coexisted in the pulmonary artery.

Etiology of Embolism.—Lotheisen [15] makes the following observations: Most emboli originate from the veins of the lower extremity. In his sixty-six cases, forty originated there.

Following parturition or gynæcologic operations, emboli have originated from thrombosis of veins about the uterus.

The hemorrhoidal plexus has also been indicted several times.

Bumm describes two cases following operation on the rectovaginal septum.

Embolism usually occurs after a previous period of rest in bed followed by sitting up, straining at stool, or muscular effort, manipulation or massage of the leg, rubbing in ointment or even the application of a bandage or zinc gelatine boot (Velpeau).

Wyder [34] saw pulmonary embolism following colpoperineal plastic.

Symptomatology of Thrombosis.—Pain, swelling, masses in superficial veins (or deep veins).

Thrombosis—sudden pulse rise with normal temperature, later temperature rise with embolism. After this temperature may descend while the pulse remains elevated (Mahler[34]).

Welch [33] gives one of the clearest descriptions of symptoms. Death may be instantaneous from syncope. More frequently the patient cries out, is seized with extreme precordial distress and violent suffocation, and dies in a few seconds or minutes. Or when there is still some passage for the blood, the symptoms may be prolonged for several hours or even days before the fatal termination. The symptoms of large pulmonary embolism are the sudden appearance of a painful sense of oppression in the

chest, rapid respiration, intense dyspnœa, pallor followed by cyanosis, turgidity of the cervical veins, exophthalmos, dilatation of the pupils, tumultuous or weak and irregular heart action, small empty radial pulse, great restlessness, cold sweat, chills, syncope, opisthotonous and convulsions. The intelligence may be preserved or there may be delirium, coma, and other cerebral symptoms. Particularly striking is the contrast between the violence of the dyspnœa and the freedom with which the air enters the lungs, and the absence of pulmonary physical signs, unless in the more prolonged cases, it be the sign of œdema of the lungs.

Wyder[24] enumerates the symptoms of pulmonary embolism as follows: (1) dyspnœa (cardiac apnœa); (2) anxiety; (3) cyanosis; (4) dilated pupils; (5) powerful and irregular heaving of heart; (6) pulse imperceptible; (7) death following syncope.

Auscultation during pulmonary embolism reveals often a blowing murmur during systole or systole and diastole at the base. Henning heard this in 4 cases out of 33.

In Lotheisen's 66 cases it was reported 3 times. Pain in the region of the scapula or in the right or left hypochondrium. Consciousness is usually retained to the last. Rarely delirium, syncope, or convulsions occur. Sense of coldness complained of; they frequently shiver.

Regarding the diagnosis, Meyer[20a] considers next to the clearly demonstrable enlargement of the right heart, the accentuation of the second pulmonary sound of importance.

Differential Diagnosis.—Wyder[24] states that atheroma of coronary arteries gives a similar picture. These patients usually give a history of previous similar attacks.

Schumacher[20] calls attention to the fact that the diagnosis of pulmonary embolism may be difficult because a suddenly occurring internal hemorrhage, also myodegeneration of the heart, can produce like symptoms.

Prophylactic Treatment.—Many suggestions have been made in the direction of prophylactic treatment, of which the following are the most important.

Wyder[24] lays down the following rules:

(*a*) In chlorosis give iron.

(*b*) In cases of pregnancy avoid operation in the neighborhood of the uterus, anus, and vulva if possible. Such operations may cause miscarriage, which aids in liberation of emboli.

(*c*) In cases of operation avoid hæmatomata, because thrombosis is readily set up in adjoining veins.

(*d*) In cases of venous stasis in the lower extremities elastic bandages and massage are recommended to avoid thrombosis. If thrombosis is suspected, however, these should not be employed (v. Jurgensen). The author condemns massage in all cases for fear of liberating emboli.

(*e*) For the same reason, Puternan goes so far as to avoid massage in all cases of fracture until three weeks after injury.

(*f*) In cases of thrombosis all movements should be avoided. Patients should not sit up in bed. Straining at stool should be avoided by the use of proper cathartics.

(*g*) Elevation of the leg has no value.

(*h*) Cold compresses changed every hour or two and laid on the anterior surface of the leg, so as not to make necessary the elevation of the leg, are advised.

Rest in bed is mandatory as long as symptoms of thrombosis or capillary thrombi are present. Even after the swelling has disappeared the patient should remain in bed for several days.

Tally recommends as a prophylactic measure that good contraction of the uterus should be sought after labor.

In endocarditis undue muscular effort, including straining at stool and severe coughing should be avoided.

Thane [37] suggests the undesirability of prolonged use of Trendelenburg position, as it may predispose to pulmonary embolism.

Ward [31] speaks of the abandonment of extensive exposure of the levator ani muscle during perineoplasty to prevent bruising of the numerous small veins exposed.

The length of time that a limb should be kept immobilized should be gauged by the disappearance of swelling, tenderness and fever which often takes one or two months. The return to normal activity should be by easy stages.

Grober [15] advises where possible the excision of the original site of thrombosis.

When thrombo-embolism occurs, keep patient absolutely at rest in bed and give morphia for distress.

Welch [32] advises the following points in the treatment of thrombosis:

(*a*) Prophylactic measures should be directed toward maintaining good nutrition, strengthening the heart's action and warding off secondary infection.

(*b*) He mentions the use of citrated milk; 20 to 40 grains of citrate to the pint in diseases conducive to thrombosis.

(*c*) Absolute rest, suitable position, and immobilization of the thrombosed extremity, and nourishing diet to ward off embolism.

Caution patient against moving the leg; palpation of affected limb should be of the gentlest sort and is better omitted altogether. The patient should not be allowed to walk in less than forty days. After the danger of embolism is passed, massage and bandaging may be employed to advantage.

Wilson [33] enumerates the following precautionary measures:

(*a*) Reduction of vascular traumatism to minimum at operation.

(*b*) The encouragement of very early free movement on the part of the patient.

(*c*) The post-operative administration of drugs to increase the coagulability of the blood is of questionable value as far as thrombosis and embolism are concerned.

(*d*) Measures leading toward the reduction of bacteræmia. Eliminate foci of infection, treat with vaccine, etc.

Bidwell [5] insists that methods of prevention are of more importance than methods of treatment, and these include the treatment of anæmia before operation, giving excess of fluids, the use of citrates, and getting the patient up as soon as possible after operation. At the same time lime salts, magnesium carbonate and milk should be avoided.

He states that the tendency to coagulation is decreased by oxygen, by improving the force of the circulation, by alcohol, by excess fluids and reduction of solids, by citric acid, rhubarb, acid fruits and wines and by tobacco.

Wyder [34] considers the most important prophylactic measures to recognize the presence of thrombosis and avoid everything which may loosen emboli.

Thorough blood study in cases of anæmia he advises to postpone operation until this is relieved. Administer iron in these cases.

To prevent femoral thrombosis after an operation Bidwell [5] believes that we should avoid the risk of injury to the edges of the wound by placing gauze pads beneath our retractors, and by using them as gently as possible; we should avoid an exclusive milk diet; we should keep the lower bowel unloaded, so as to minimize the interference with the blood-stream through the common iliac vein by pressure from the sigmoid; we should give excess of fluids, especially by rectum, and we should avoid calcium salts and carbonate of magnesia. The patient should have plenty of fresh air and should be given citrates, if milk is allowed.

Th. Kocher agreed with Volker [20] and went still further, saying that he sought for locations where there may have been old thrombi, *e.g.*, in the varices of the leg. He thinks it clearly indicated to cure such varices before subjecting the patients to major

operations during which they may succumb to embolism. For this purpose he makes use of ligatures of the large saphenous by Trendelenburg's method by multiple intermediate ligations of the varices.

Playfair [28] agrees that the main element in the treatment of such cases is the most rigid rest and a nourishing supporting regimen.

Paul Zweifel [27] reports eighteen pulmonary thrombosis deaths in 1832 cases operated upon a table interfering with the veins of the lower extremities and only three in 860 cases operated upon a table which did not have this feature. In the former series, the deaths from this cause amounted to one in 100; in the latter series, one in 286.

He advises the following precaution which, according to his enormous clinical experience, will greatly reduce the occurrence of pulmonary thrombosis. Avoid all pressure upon the veins of the lower extremities, such as occur from permitting the legs to hang over the lower end of the table in operations in the Trendelenburg position with the use of the tables which drop the lower end.

He gives further statistics, according to which he lost five cases from thrombosis in 450 abdominal sections; three in operations for uterine fibroids, one for carcinoma of the uterus, and one for extirpation of a cyst in a patient seventy-five years of age. Following this high mortality, notwithstanding the fact that the extremities were not traumatized during the operation, a change in the technic was followed by only five deaths from thrombosis in 2060 laparotomies and only one in 484 operations for uterine fibroids, and this in a patient who had suffered eight weeks previously from a cerebral embolus. All the five deaths mentioned occurred in decrepit, anæmic, or cachectic patients.

The change in technic consisted in the absolute control of oozing of blood and in applying a purse-string suture covering absolutely all raw surfaces in the pelvis.

R. Olshausen [25] attributed the frequent occurrence of pulmonary thrombosis to the use of an operation table which caused the knees to be flexed in placing patients in the Trendelenburg position, causing a compression of the veins of the lower extremities; 2443 with 14 pulmonary thromboses; of these there were 571 uterine fibroids, with seven thromboses. Following these cases he reported fibroids with no thromboses in cases operated on a table without compression of the veins of the legs.

Therapeutic Treatment.—According to Wyder,[24] upon the occurrence of pulmonary embolism rapid therapeutic measures are necessary.

1. Subcutaneous injections of ether and camphor in oil. Von Kenezy gave ether injections hourly for two days and claims that the patient felt definite alleviation after each. Oeder gave .2 gram camphor every five minutes or 2.4 grams in one hour, and claims a recovery with this. Caffein and digitalis may be used intravenously.

2. Morphia .02 gram, used by most clinicians, counteracts shock, and if death supervenes, makes this less painful.

After infarction combat dyspnœa and distress with opiates.

Lead acetate may be administered. He does not hold artificial respiration useful, because death does not occur from respiratory obstruction but rather lack of oxygen in the blood.

Bidwell [5] states with regard to treatment, while he recognizes that little can be done when the whole of the right pulmonary artery is blocked; oxygen, strychnine, and saline injections are always given, and in one case life was prolonged fifteen hours.

Church [8] urges the use of oxygen gas inhalation for these patients on the basis of its demonstrated value in the treatment of pneumonia.

Meyer [29a] describes the removal of pulmonary emboli by Trendelenburg's operation in detail. Trendelenburg performed twelve operations of this kind on man without a permanent recovery.

He states that the coagulation of blood and recurrence of embolic accident can be avoided by injection of hirudin.

He distinguishes three classes of pulmonary embolism:

1. The one causing immediate exitus which evidently is due to shock, as not infrequently only a partial thrombosis of the artery may be found at autopsy.

2. The one causing death within a few minutes. Here thrombosis is perfect, separating pulmonary and greater circulation. The right heart becomes quickly overdistended.

3. The one of protracted course which is more frequently observed. Here one of the main branches of the artery and subdivisions become suddenly clogged, only gradually total obstruction sets in.

As a matter of fact, only the third category will furnish cases with indication for operation and the task of establishing this indication is rendered difficult on account of the experience that some of these patients get better under conservative treatment. He considers operation imperative if medical treatment fails to bring improvement.

Four times this operation was performed at the Zurich Clinic, but the patients could not be saved.

TABLE OF CASES OF DEATH DUE TO PULMONARY THROMBOSIS DURING THE FIVE YEARS 1915 TO 1919 IN THE AUGUSTANA HOSPITAL

	Date	No.	Sex	Age	Diagnosis	Operation	Operator	Post-operative days	Predisposing cause of embolism
1	7/15/15	43793	M	42	Osteochondroma of left ileum	Excision of osteochondroma	A. J. O.	4	Phlebitis.
2	9/ 4/15	44273	M	46	Gastric ulcer; cholecystitis, chronic appendicitis	Posterior gastro-enterostomy cholecystectomy; appendectomy	N. M. P.	6	Perforated ulcer with preoperative loss of weight.
3	4/13/16	46790	F	33	Cholecystitis, retroversion, rectocele	Cholecystectomy perineorrhaphy	N. M. P.	2	
4	7/ 7/16	47388	F	44	Carcinoma of rectum	Colostomy	A. J. O.	3	
5	5/21/17	50718	M	65	Hypertrophied prostate	Suprapubic prostatectomy	N. M. P.	15	Cachexia; phlebitis left leg.
6	6/12/17	50802	F	33	Gestation; 3rd degree laceration perineum	Parturition perineorrhaphy	R. H.	14	Perineal sepsis.
7	4/28/18	54315	F	51	Cholelithiasis; carcinoma of rectum	Cholecystostomy panhysterectomy	A. J. O.	7	Anæmia; cachexia.
8	12/16/18	56932	F	43	Floating kidney; cholecystitis; appendicitis	Nephropexy; cholecystotomy; appendectomy	A. J. O.	12	Preoperative loss of weight, 35 pounds.

Year	Operations	Obstetrical deliveries	Laparotomies	Hysterectomies
1915	3,665	153	1204	94
1916	3,510	187	1218	88
1917	3,303	211	927	133
1918	3,303	278	871	107
1919	2,915	270	1055	106
	16,696	1099	5275	528

Seven deaths in 16,696 operations = 1 case in 2385 = 0.042 per cent.
One death in 1099 obstetrical cases = 1 case in 1099 = 0.1 per cent.
Five deaths in 5275 laparotomies = 1 case in 1055 = 0.1 per cent.
One death in 528 hysterectomies = 1 case in 528 = 0.5 per cent.

We have reviewed the histories of all cases of death due to pulmonary thrombosis which occurred during the past five years in the Augustana Hospital following surgical operations, including the years 1915 to 1919,

eight cases. These are represented in the accompanying table. Of these, four cases occurred in my own service, three in the service of my colleague, Dr. N. M. Percy, and one in the obstetrical service in a patient in which a very extensive perineal laceration had to be repaired immediately following delivery. It is likely that the thrombosis had no relation to the operation, because deaths from this cause have occurred many times after delivery of cases in whom no surgical repair was made, but in order to make our statistics complete the case had to be included.

During these years 16,696 operations were performed in all in the Augustana Hospital with 5275 laparotomies, and of these 528 were hysterectomies which in all other statistics we have encountered showed the largest percentage of deaths from pulmonary thrombosis.

During the same period 76 cases of so-called pernicious anæmia were operated for splenectomy, cholecystectomy and appendectomy in patients who, according to all statistics, should have resulted in a considerable number of deaths from pulmonary thrombosis. Among these cases, most of which were operated by my colleague, Dr. N. M. Percy, there occurred no deaths from this cause.

In every case, however, the operation had been preceded by one or more transfusions of whole blood without the addition of citrate of soda.

Recognizing the etiological importance of the twelve conditions mentioned at the beginning of this paper, whose validity seems to have been established by many authorities noted for their keenness of observation, it would seem worth while to investigate whether death from pulmonary thrombosis could have been avoided in any or all of these cases had every possible precaution been taken to eliminate each one of these etiologic factors to the greatest possible extent.

1. *Local Infection.*—It seems certain that even the slightest amount of local infection may cause a thrombosis in a neighboring vein which may be loosened and serve as the cause of a fatal pulmonary thrombosis when it becomes lodged in the pulmonary vein. Although there is no evidence in any one of our cases that this has actually occurred, yet it seems important to still further perfect aseptic methods of operation. Of course, hæmostasis must be accomplished as a result of a normal thrombosis of the ends of the cut vessels, but it does not seem likely that such a thrombus will ever become loosened, so that it can cause death due to pulmonary thrombosis.

2. *Anæmia.*—Most of the patients, especially Cases II, V and VII, showed some degree of anæmia. It seems likely that it might have been possible to correct this by more careful preliminary treatment or by transfusion of whole blood.

3. *Slowing of Blood Stream.*—It has been claimed that keeping patients for a long continued operation in the Trendelenburg position would interfere with the blood stream in the extremities to such an extent that this serves as a predisposing cause. In none of our cases, except

Case II, could this have been the cause, as none of the other operations were of long duration.

4. *Subnormal General Physical Condition.*—This obtained in Cases II, VII and VIII, but aside from the possible improvement which could have been secured by the preliminary transfusion of whole blood, it is doubtful whether this cause could have been eliminated to a marked extent.

5. *Cachexia.*—The only treatment which could be of any benefit would again be the transfusion of whole blood. Two of our own cases belonged to this group, and were similar cases to come under our care in the future, we should make use of prophylactic transfusion of whole blood.

6. *Microörganisms in the Blood.*—None of our cases belong to this group, but we believe that in several cases of this class we have obtained great benefit by the transfusion of whole blood, although it is, of course, impossible to state that thrombosis has been prevented in any given case, although it failed to appear in any of these following transfusions.

7. *Excess of White Blood-cells.*—This cause did not exist in any of our cases.

8. *Inefficient Hæmostasis.*—Until very recently we had not fully appreciated the importance of this etiologic factor, and it is quite possible that some of our deaths may have been due to an error in this direction, although fortunately our method of closing the stump in hysterectomy for many years has corresponded to that upon which Zweifel[37] lays so much stress, and it is possible that we may have escaped many deaths from pulmonary thrombosis in this class of cases without being entitled to any credit for this.

9. *Traumatization of Tissues with Retractors, Etc.*—It seems likely that we have not exercised proper care in this direction, because until recently we have made use of heavy retractors for holding open the abdominal wound. These have now been discarded, and although we cannot trace any case directly to this cause, it seems likely that our technic has been bad in this direction.

10. *Injury to Veins of Extremities Due to Badly Arranged Operating Table.*—Zweifel and others have traced a number of their cases directly to the use of a table in which the knees are bent, so that the veins are compressed during the operation. They found a marked reduction of fatal cases upon abandoning this particular table. We have never used this type of table, but it seems proper to again direct attention to this apparent cause.

11. *Injury to Intima of Veins.*—Rough handling of tissue in the vicinity of the wound undoubtedly often causes an injury to the intima of veins, and it seems worth while to train one's self and one's assistants to avoid this as well as all other forms of unnecessary traumatization of tissues.

12. *Excess of Calcium Salts in the Blood.*—So many authors mention this as an etiologic factor that it may be important to consider it. So far we have paid no attention to this element.

It seems likely from our observations that in the future we will be justified in systematically adding the transfusion of 600 c.c. of whole blood to our preliminary treatment in a considerable proportion of cases belonging to a class which has in the past made up our list of deaths from pulmonary thrombosis and that the other precautions will be carried out with greater care.

It is likely that this will result in a considerable reduction in the death rate from post-operative pulmonary thromboses.

A short analysis regarding the etiology of the thrombophlebitis as shown in our eight tabulated cases is as follows:

CASE I.—A man, forty-three years of age, who had an enchondrosteoma 21 by 11 by 7 cm. removed from the lateral surface of the left ileum. The wound was aseptic and there is nothing in the history which could serve as a predisposing factor, except the fact that ten years previously the patient had suffered from thrombophlebitis of the veins in both legs and that the venous circulation had been impaired ever since this occurrence. The wound was aseptic, but his thrombophlebitis must have lighted up again. Had this patient received careful preliminary treatment for several weeks, it is quite likely that the thrombophlebitis might have been prevented.

CASE II.—A man forty-five years of age, giving a history of gastric ulcer complicated with cholecystitis and chronic appendicitis, had lost considerable in weight and was anæmic as a result of loss of blood from his gastric ulcer. The gall-bladder was thick, universally adherent, sacculated and filled with black viscid bile. The mucosa was granular. This gall-bladder was removed. The appendix was sacculated and universally adherent. In this case, the patient died six days after the operation from pulmonary thrombosis. This patient had anæmia and mild cachexia, also a great amount of disturbance because of the extensive operation and extensive adhesions. The operation lasted one hour and a half, consequently several factors were present which would account for the pulmonary thrombosis. A preliminary transfusion of whole blood and performing the operation in two stages, a gastro-enterostomy at the first stage and a cholecystectomy and appendectomy after the patient had fully recovered from the first operation, would probably have prevented the unfavorable result.

CASE III.—A woman, thirty-three years of age, showed nothing in her history which would predispose to the development of thrombophlebitis. She had a normal cholecystectomy for the relief of gall-stone disease and perineorrhaphy performed with an anterior suspension of the uterus. She died from pulmonary thrombosis two days following the operation. It seems likely that some of the veins in the vicinity of the broad ligaments or rectum must have been injured during the operation. The case emphasizes the importance of protecting the veins.

CASE IV.—A woman, forty-four years of age, with carcinoma of the rectum. This patient showed but very slight cachexia and was otherwise in fair condition. A simple colostomy was performed. The patient was under the influence of the anæsthetic less than half an hour, and there was nothing that one would reasonably expect to cause a thrombosis except that the mesentery of the sigmoid was transfixed and a strand of gauze and glass tube were passed through to keep the intestines from slipping back into the abdominal cavity. Evidently, a thrombus was loosened from one of these veins. Whether preliminary transfusion of whole blood might have prevented this, it is difficult to say.

CASE V.—A patient sixty-five years of age had a simple suprapubic prostatectomy from which he made a very satisfactory recovery for fifteen days, when he died suddenly from pulmonary thrombosis. On the afternoon of the fourteenth day a phlebitis developed in the femoral vein, which undoubtedly accounts for the embolus which gave rise to pulmonary thrombosis.

CASE VI.—A patient, thirty-three years of age, who suffered from a severe laceration of the perineum during delivery which was repaired immediately, died fourteen days later from pulmonary thrombosis. In this case there was severe suppuration of the perineal wound. Whether the infection from this source was responsible for the pulmonary thrombosis or whether the latter condition resulted from a loosened thrombus from the uterus, we have been unable to determine.

CASE VII.—A patient, fifty-one years of age, suffering from gall-stones and cancer of the rectum with marked anæmia and cachexia, died of pulmonary thrombosis seven days after the operation, which consisted of a cholecystectomy and panhysterectomy. In this patient the anæmia should have been corrected by transfusion of whole blood and panhysterectomy should have been performed at the first operation and cholecystectomy after the patient had regained strength.

CASE VIII.—A patient forty-three years of age, suffering from a floating kidney which could be moved over the greater portion of the abdominal cavity, from cholelithiasis and appendicitis. She had lost 35 pounds in weight previous to the operation. The operation performed consisted of cholecystostomy, appendectomy, and suturing the kidney in place. The gall-bladder contained a large number of stones. In this case, good judgment would have undoubtedly prevented the pulmonary thrombosis, because with the patient in this condition, the appendix and gall-stones should have been removed first and the floating kidney operated at a second operation.

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CORRIGENDA

MAYO ON THE CALLOUSED ULCER OF THE POSTERIOR WALL OF THE STOMACH.

In the Annals of Surgery for July, 1920, page 109, 3d line from the top, 3.2 per cent. instead of 4.7 per cent. as the average mortality in the Mayo clinic of operations for gastric ulcer.

THE CALLOUSED ULCER OF THE POSTERIOR WALL OF THE STOMACH*

By William J. Mayo, M.D.
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From July 1, 1914, to July 1, 1919, 647 operations were performed in the clinic on 638 patients with gastric ulcers, with an average operative mortality of 4.7 per cent. All patients who died in the hospital following operation are statistically classified as having died from operation, without regard to the cause of death or length of time after operation. During this same five-year period 2734 operations were performed on 2720 patients with duodenal ulcers, with an operative mortality of 1.2 per cent.

In twenty-eight of the series of 638 gastric ulcers the ulcers were multiple. Five hundred thirty-four were located on or around the lesser curvature of the stomach, eighty-five involved the posterior wall, nine were on or around the greater curvature, five were on the anterior wall, and five were not located exactly because of the emergency nature of the operation.

Ulcers of the Posterior Wall of the Stomach.—In dividing the stomach into thirds (Fig. 1), the pyloric third showed only 8.2 per cent. of ulcers of the posterior wall, the middle third the great preponderance of 75.8 per cent., and the cardiac third 16.5 per cent. The ulcer crater varied from 2 cm. to 11 cm. in diameter. All the ulcers were of the chronic perforating varieties; in fifty-three cases the coats of the stomach were penetrated and the crater of the ulcer encroached on a neighboring viscus, usually the pancreas; a few also encroached on the liver, or the transverse colon, or were imbedded in a mat of adhesions. Fifty-seven of the eighty-five patients with ulcers of the posterior wall of the stomach were males and twenty-eight were females. The average age was forty-three and nine-tenths years; the oldest patient was sixty-nine and the youngest eighteen years. The average duration of symptoms was six years and six months. The average weight loss was 17.8 pounds, the greatest loss 39 pounds, and the least 3 pounds. Fourteen patients had marked secondary anæmia, and anæmia was present in all, often accompanied by a cachexia. The maximum hæmoglobin was 66, the minimum 26. The pain was in the epigastrium in fifty-six patients, in the back in twenty-four; it radiated to the right in fifteen cases, to the left in eight, and downward in eight; in eleven it was given indefinitely as in the stomach and mid-abdomen. Food gave relief in fifty cases. The obstruction was not extreme in any case, although distinct lagging of food or obstruction varying from slight to moderate was demonstrated in 35 per cent. of the series.

Hour-glass stomachs were excluded from the series. Acids averaged about normal for the age, and there was little difference between the acid

* Read before the American Surgical Association, May 3, 1920.

(five points above) of the patients who had hemorrhages and those who did not. Nineteen patients (20 per cent.) had gross hemorrhages; seventeen vomited blood, and twelve, whether or not they vomited blood, had blood in the stools. The clinical diagnosis was correct in seventy-one cases. Cancer was diagnosed in six cases, pyloric obstruction in two, gall-stones or gastric ulcer in four, and duodenal ulcer in two. The X-ray diagnosis was correct in seventy-one cases; duodenal ulcer in two, and " negative stomach " in ten. No X-ray was made in two cases. The ten cases in which the diagnosis was negative represent a type which is now better understood. Within the last three years the X-ray technic has improved and diagnosis by this means is established in above 90 per cent. of lesions of the stomach.

To epitomize: The main characteristics of ulcer of the posterior wall of the stomach are: Chronicity, lesions usually large, more or less continuous distress, occasional exacerbations from localized peritonitis, and anæmia often accompanied by marked cachexia.

Even at operations it is sometimes difficult to determine whether the condition is ulcer or cancer until the lesion is actually exposed and a specimen secured for immediate microscopic examination. In our early experience in some cases clinically diagnosed as ulcer in which gastroenterostomy only was done, the patients died later from carcinoma of the stomach, but at a time remote from the operation. It must be admitted that this is an impressionistic view because there was no specimen removed for evidence that the growths in question were not cancerous at the time the operations were performed. But since the ulcers were not removed, why did the patients live so long before the cancers became manifest? The opposite mistake certainly was made, because in some cases (7) that were clinically diagnosed cancer and in which gastroenterostomy was done, the patients lived too long after the operation for the original diagnosis to have been correct. And here is just the difficulty in trying to settle the question of the frequency with which chronic ulcers of the stomach undergo malignant degeneration. Clinical diagnosis is notoriously defective, post-mortem evidence cannot prove the original disease, and operations that do not permit the actual excision of the lesion or the removal of a specimen for microscopic examination are open to objection. No matter how we view the question *a priori*, however, the experience of Robson, Moynihan, Pouchet, Deaver, and others, and the experience in our clinic is too large for the data to be controverted by the opinion of clinicians who have not had specimens removed and accurately examined during the life of the patient. Aschoff very properly points out that if the lesion is cancer originally the base of the ulcer will prove to be cancer. Wilson and MacCarty have shown in our cases of cancer on ulcer that cancer existed in the overhanging margin of the ulcer and not in the base.

An interesting side-light is thrown on the problem of peptic ulcers by

Balfour's investigation of the frequency of hemorrhage following operation for ulcer of the duodenum and stomach. He found, in cases of duodenal ulcer in which nothing but gastro-enterostomy was done and in which there had been hemorrhage before operation, that 1 in 8 had hemorrhage afterward, although all the other signs and symptoms were abated. In cases of gastric ulcer, however, hemorrhage occurred in only 1 in 12 following operation. Balfour explains this discrepancy as due to the fact that gastric ulcer, because of its carcinoma liability, was subjected to radical removal, while duodenal ulcer, having little or no cancer liability, was not usually excised. Balfour found that if duodenal ulcers were excised the liability to secondary hemorrhage disappeared and the mortality of cautery excision with gastro-enterostomy was not greater than gastro-enterostomy without excision. That all varieties of gastric ulcer are more serious than duodenal ulcers has been shown by data compiled from our cases by Hunter, Actuary of the New York Life Insurance Company; the death rate from duodenal ulcer in the first four years after operation was practically the same as the normal, as a matter of fact, better, while for gastric ulcer the average death rate in the four years was three times normal. A study of these tables cannot fail to leave a well founded suspicion that at least a minority of the patients died from cancer of the stomach.

Summary of Results in 85 Cases of Calloused Ulcer on the Posterior Wall of the Stomach in Which Operation was Done from July 1, 1914, to July 1, 1919.

		Per cent.
Cases	85	
Operations	87	
Deaths in hospital	4	4.7
Deaths after leaving hospital	10	11.7
Living patients reporting their condition	43	50.5
Improved	14	32.5
Unimproved	3	6.9
Cured	26	60.4
Patients not located	28	32.9

A. Cautery or Knife Excision.—Cautery alone or knife excision alone has a limited field of usefulness in cases of small ulcers in all situations. It has given good results when care is exercised not to disturb the nerve supply and the muscular efficiency. Opening the stomach and applying the cautery to the base of the adherent ulcer is not a sound procedure.

B. Gastro-enterostomy.—At times the general or local condition of the patient may indicate posterior gastro-enterostomy, or the extent of the posterior lesion may warrant anterior gastro-enterostomy.

C. Resection.—Resection of the pyloric half of the stomach by the methods of Billroth or the Polya-Balfour gives good results in suitable cases, and is the operation of choice when the pyloric region is involved. But when the ulcers lie high on the body of the stomach the operation removes a large part of the uninvolved organ that is capable of good

function. Resection in continuity (sleeve resection) of the ulcer-bearing area of the stomach, if the ulcer is in the middle third, is an excellent method, as has been shown by our experience and corroborated by Stewart. A circular piece of the stomach, including the ulcer, is removed and the proximal and distal parts of the stomach are united end-to-end with catgut.

D. Excision and Gastro-enterostomy.—After a good specimen has been secured for microscopic diagnosis the ulcer is excised with the cautery, the defect is closed with catgut sutures, and a posterior gastro-enterostomy performed. Excision of the ulcer and gastro-enterostomy would seem the logical procedure in the average case, but experience has shown that the method may fail to give complete relief if the ulcers are on the posterior wall of the stomach, because the reformation of crippling adhesions that immoblize the posterior wall sometimes follow.

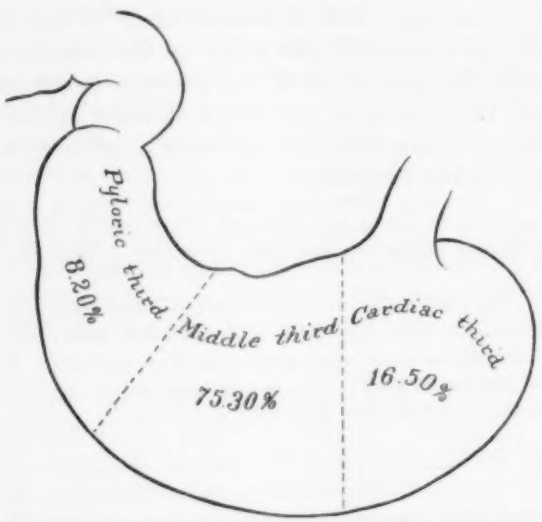

FIG. 1.—Diagram of location of ulcers found on the posterior wall of the stomach, at operation, from July 1, 1914, to July 1, 1919.

OPERATIVE TECHNIC

The procedure herewith described combines excision of the ulcer and gastro-enterostomy and is satisfactory at least in that it usually prevents subsequent posterior fixation of the stomach. The approach to the posterior wall of the stomach can be made either from above or below. I have tried both, and perhaps because I have had a larger experience with the upper approach I prefer it.

The gastrohepatic omentum is divided (Fig. 2), if necessary the gastric artery is tied, and the adhesions high on the lesser curvature are separated to secure adequate operating space. After all the adhesions are cleared away I insinuate my finger around the adherent ulcer. This

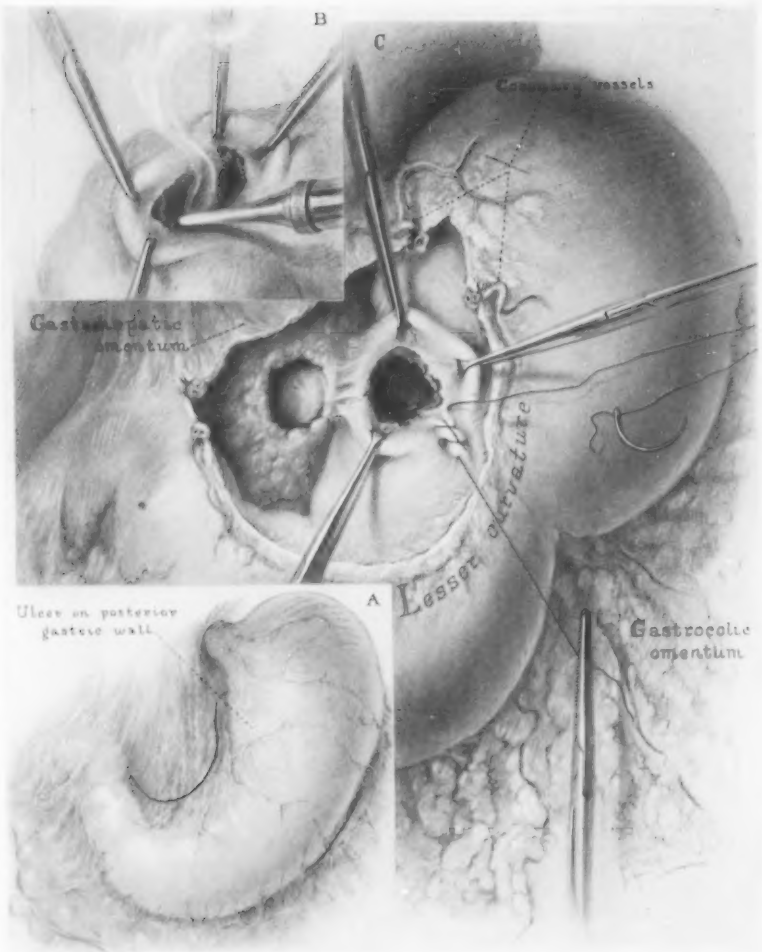

FIG. 2.—A. Lines marking proposed incisions in gastrohepatic omentum for exposure of ulcer which is removed for microscopic examination, and in gastrocolic omentum to draw the left-hand portion of the omentum proper into the lesser peritoneal cavity. B. Excising the ulcer with cautery. C. Crater of the ulcer on the posterior wall of the stomach separated from the pancreas for three-quarters of its extent, thereby exposing ulcer cavity in the pancreatic surface.

FIG. 3.—A. Ulcer being sutured transversely and omentum drawn behind the stomach covering the pancreatic incision and operative field. B. Operation completed.

hooks the involved stomach and pancreas in such manner that they can be drawn up into the wound and exposed. The stomach and its posterior attachments are held up by the finger, or by a gauze tape, and the ulcer is shaved off from the pancreas deep enough to include all the base. Sometimes in the huge ulcers the pancreas cannot be sufficiently exposed for the safe excision of the entire base of the ulcer. In such cases the pancreatic defect is carefully seared with the cautery. I have never seen fat necrosis or any harmful evidence of pancreatic leakage follow these manœuvres. The margin of the ulcer is caught with forceps in order to further its exposure. If the stomach contains a considerable quantity of fluid which has not been removed by the preliminary use of the stomach tube, the fluid should be removed by suction. Frozen sections of the base of the ulcer, of the involved pancreas, and of the margin of the ulcer area in the stomach are subjected to microscopic examination, and after this the ulcer is excised with the cautery. The posterior wall of the stomach above the ulcer will be found dilated and pouched, and the gap in the stomach is easily closed with through-and-through catgut sutures, bringing the cauterized margins of the stomach directly into contact. A second row of catgut sutures turns this line in. The direction of the suturing which prevents narrowing naturally suggests itself (Fig. 3). An opening is then made below the greater curvature through the gastrocolic omentum, and the tip of the omentum is drawn upward behind the stomach and fastened in a manner to cover the whole of the operative field. This insures speedy union and permanently separates the posterior wall of the stomach from the pancreas and liver. A posterior gastro-enterostomy completes the operation. Even if the field of operation is considerably soiled I make a proper toilet and do not use drainage; I have had no occasion to regret the omission.

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FAILURE OF PRIMARY ROTATION OF THE INTESTINE (LEFT-SIDED COLON) IN RELATION TO INTESTINAL OBSTRUCTION *

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Whatever be the cause of the inhibition of the primary rotation of the mid-gut it is perfectly obvious that when this condition is accompanied, as is usually the case, by failure of fixation of the mesentery, the conditions are ideal for the production of volvulus of large portions of the intestine. In this condition, which for brevity's sake is often called "left-sided colon," the mesentery on the right side ordinarily does not adhere to the posterior peritoneum. Why, we do not know, unless it is because we have to do with the mesentery of the small intestine.

In the normal condition of rotation of the intestine when the hepatic flexure of the colon grows over to the right or is pushed over by growth of that portion of the transverse colon which lies to the right of the duodenum, the superior mesenteric artery through its large branch which becomes the middle colic artery furnishing a relatively fixed point, the large intestine itself generally adheres on the right side, though not so uniformly as the descending colon adheres on the left side, and it is not unlikely that adhesion of the intestine occurs before that of the mesentery. At least the irregularity of the mesenteric adhesion leaving, as it often does, crypts and fossæ which are sometimes of surgical importance in relation to internal hernia, suggests that the intestine adheres first. The mechanism of this adhesion is also unknown. It is hardly competent to say that it is congenital. The fine cicatricial bands which are commonly observed about the ascending and descending colon suggest that the cause of the adhesions is some pre-natal inflammatory process, pre-natal peritonitis, if you will. Possibly the large bowel is less actively mobile and more penetrable by irritating substances. Certainly there is much material absorbed by the colon in pre-natal life desiccating the fluid contents of the small bowel into the stiff paste of meconium which is retained for a long time in the colon. If a pre-natal inflammation of the outer wall of the large bowel is presupposed it would readily enough account for these adhesions and other congenital bands which are frequently found. Such explanation has been offered for the production of this most interesting congenital anomaly, the failure of rotation of the mid-gut. An example of an adventitious band occurred in the second case here reported, where there was found a cord-like mass of tissue leading from the posterior wall of the abdomen to the posterior surface of the cæcum which lay in the right iliac fossa notwithstanding the fact that the small intestine lay completely to the right of the colon. The band was

* Read before the American Surgical Association, May 3, 1920.

manifestly the result of adhesion of the cæcum. Such a band of adhesion would furnish perfect conditions of obstruction by herniation of loops of bowel through the opening between it and the mesentery, but also if it were accompanied by "left-sided colon" with failure of attachment of the mesentery it would furnish a fixed point about which the entire intestine might be twisted, making a double volvulus—right and left.

Under normal conditions that part of the intestine between the lower part of the duodenum and that portion of the transverse colon which is

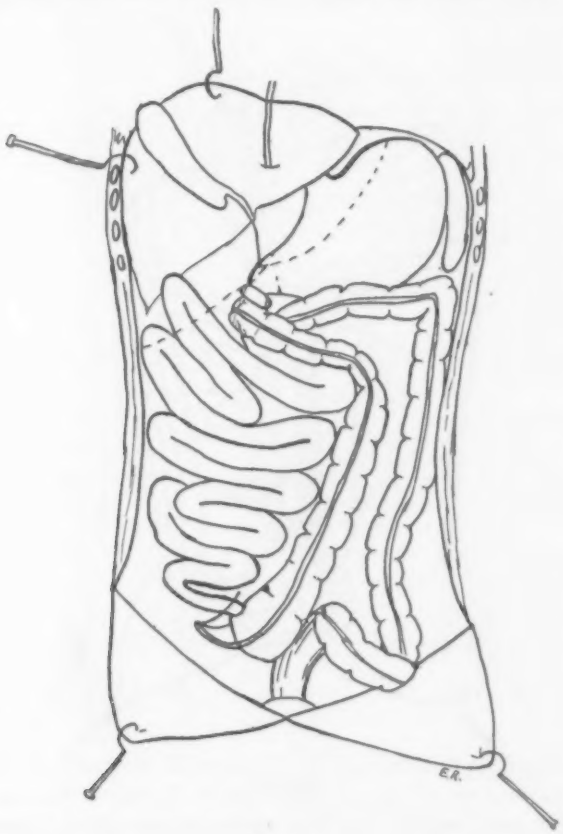

FIG. 1.

supplied by the mesocolic artery rotates to the left, *i.e.*, so as to form a left-handed spiral at the point of crossing of the large and small intestines, throwing the duodenum to the left beneath the superior mesenteric artery, so that afterwards the artery is found to pass down directly over its anterior surface. The angle of this left-handed rotation is said to be 180 degrees, but, as a matter of fact, it is 270 degrees or more, for in early embryonic life when the intestine is attached to the umbilicus by the vitelline duct the mesentery lies in the sagittal plane. As the gut grows and the loop of the small bowel and cæcum become drawn out and rotation

takes place, as it were, about the vitelline duct or Meckel's diverticulum as an axis, what was originally the left side of the mesentery becomes not merely the right side (rotating 180 degrees, which would place the large bowel simply in front), but by further rotation of 90 degrees becomes the posterior layer, *i.e.*, it adheres to the posterior layer of the peritoneum and lies in the coronal plane or beyond.

It is said that bands of adhesions are sometimes the effective cause of the inhibition of rotation, but of the three cases of "left-sided colon" here reported such were not made out in the first case, although a most

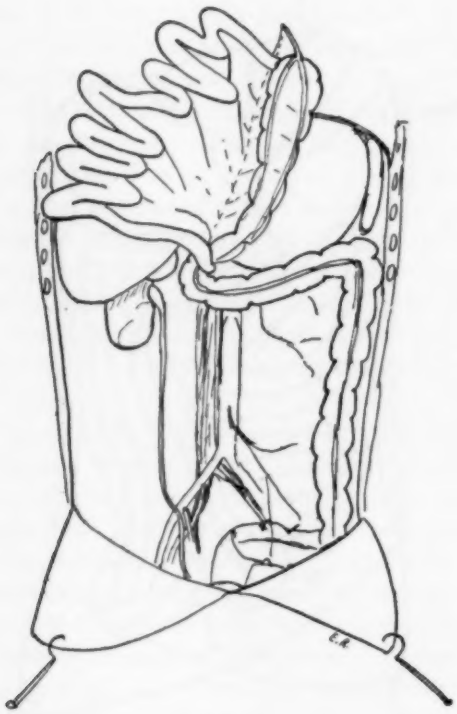

FIG. 2

careful autopsy was made. In the second case observation is simply lacking, since in the hurry of a desperate operation there was neither time nor justification on other grounds for extended search after interesting phenomena. In the third case, however, there were very definite bands, one on each side, nearly an inch wide, consisting of simple fibrous tissue which if of inflammatory origin must have been very old, even pre-natal, for they presented no evidence of inflammatory reaction, no thickening, no cicatricial contraction, no adventitious blood-vessels. Had they been the result of bacterial inflammation secondary to the volvulus they would almost certainly have contracted sufficiently to have caused strangulation of the intestine or fatal obstruction. But admitting that these bands were

the cause of the failure of rotation of the intestine there is still a question as to their origin.

In the three cases here reported, all that I happen to have seen of "left-sided colon," the condition was found at operation for intestinal obstruction. In the first case the obstruction was not the result of the congenital anomaly, but was due to carcinoma of the transverse colon. In the other two the failure of rotation of the gut and fixation of the mesentery was the predisposing if not the direct cause of the obstruction.

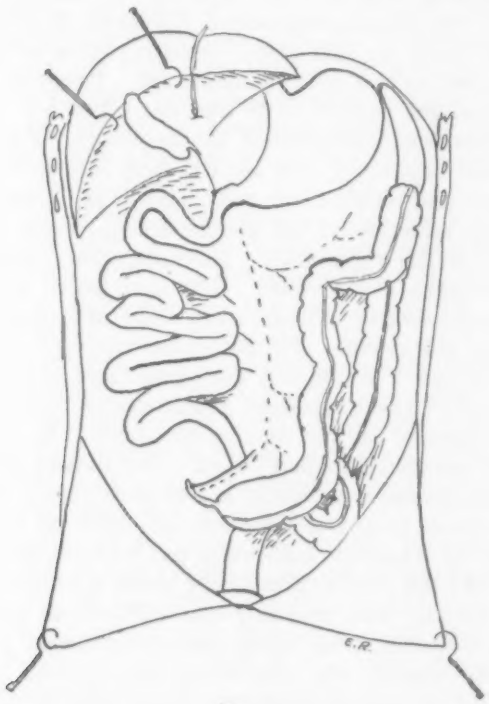

FIG. 3

CASE I.—A man of sixty-two years, merchant of Seward, Alaska, who had always enjoyed rugged health, came to operation for obstruction of the transverse colon which had been progressive for a year. The distention was greatest on the right side and in the upper abdomen. Incision for a proposed right inguinal colostomy showed the right flank filled with distended hypertrophied coils of small intestine, the cæcum not to be found, a large, hard, fixed tumor in the region of the transverse colon, liver filled with metastases, an immense elastic coil of distended colon on the far side of the small bowel, evidently the ascending colon. The hepatic flexure was successfully brought to the lateral abdominal wall in front of the small bowel, sutured into the incision and opened some hours later. The patient escaped peritonitis but succumbed on the fourth day to broncho-pneumonia. Autopsy showed "left-sided colon," no cicatri-

cial or other bands about the duodenum or jejunum, carcinoma of transverse colon involving the mesentery and invading the portal vein with shower of metastases in the liver.

In brief, the "left-sided" colon in this case in no wise interfered with a long and active life, but the unusual and not to be anticipated location of the colon with reference to the distended loops of small intestine added much to the difficulties of the operation for relief of the obstruction.

CASE II.—A woman of forty-one years, mother of one child, had always been strong and well till depleted by hemorrhage from a large myoma. Hysteromyomectomy rendered difficult by extensive adhesions from an old pelvic inflammation entailed considerable traumatism to the lower peritoneum. Because of the weakened condition of the patient time was not taken for exploration of the upper abdomen nor removal of the appendix. Patient made an excellent primary recovery, but on the fifth day developed a temperature of 102° and on the sixth 105°, pulse 124, leucocytosis, vomiting, evidence of infection of the uterine stump, abdominal distention more marked on the right side. Enemas, hot compresses, withdrawal of food gave much relief. The distention which remained was thought to be due to inflammatory ileus or to obstruction from adhesions about the cæcum. Patient so far recovered as to be able to take food for about a week, but the symptoms of obstruction became more marked and secondary septic symptoms coming on the abdomen was opened on the nineteenth day. The distention was entirely in the cæcum and small bowel. The cæcum was adherent to the anterior abdominal wall where it had perforated. The appendix which was 6 inches long was inflamed and adherent by its tip to the uterine stump. It was first removed to get it out of the way and then it was found that the obstruction of the colon was due to volvulus, a right-handed twist. The twisting was difficult to explain because the relations of the terminal ileum and cæcum were normal and the mesentery of the cæcum was attached to the posterior abdominal wall by a cord nearly an inch in diameter and 6 inches or more long. The rotation in the volvulus, therefore, could only have occurred by the gut (cæcum and portions of the small intestine) passing around itself as an axis, the mesenteric cord being a sort of pivot, at a time anterior to the attachment of the appendix to the uterine stump. The most likely explanation is that in withdrawing gauze pads which were used to hold the intestines back in the first operation, they rubbed or pulled on the bowel sufficiently to give it the right-handed twist. When the volvulus was untwisted the large bowel was found to be necrotic in spots and had to be resected. There were embarrassments in making the necessary lateral anastomosis, for the ascending colon was in the pelvis and adherent and led upward toward the splenic flexure on the left side of the small intestine. Anastomosis was accomplished and patient made a normal recovery. She was well when last seen, some six years later.

CASE III.—A boy of five years, whose history from birth was that

of a delicate child who suffered from early infancy from recurring attacks of colicky pain and vomiting. His nutrition was always the greatest concern of his parents, but he managed to grow somewhat in spite of his starvation. He was bright mentally and had learned to refrain from eating whenever he felt one of these attacks coming on, and that was pretty frequently, for when the stomach was empty the attacks sooner subsided. He had never known what it was to have a full meal. When he came under our care he was just recovering from his severest attack of obstruction. He was far below normal in weight, had almost no fat; his face was pinched and purplish, and his extremities blue and cold. He was too weak to be up more than a short time each day. Loth to move, he was quite content to lie in bed and watch other children play. The abdomen was distended above, but was concave below, the abdominal wall tense as in the atrophy of starvation, but not spastic. No tumor could be made out. No icterus.

From the history and the findings it was evident that the child was suffering from some form of chronic obstruction high up in the intestine, not complete, but probably due to some congenital anomaly. We thought possibly congenital stricture of the duodenum or hypertrophic pyloric stenosis. After administering fluids by rectum we opened the abdomen and found the explanation of the obstruction in a volvulus of the intestine at a point just below the biliary papilla. Included in the volvulus was the entire small intestine, the ascending and part of the transverse colon. Only the stomach, pylorus, and upper duodenum were distended; the remainder of the intestine was collapsed.

The entire mass of the intestine, having no other attachment than at the point of the twist, which consisted of a cord hardly more than an inch in diameter, was lifted bodily out of the abdomen. The posterior peritoneum was continuous from the descending colon across to the right side of the abdominal wall, and because of the absence of fat was transparent, so that the right kidney and ureter, the aorta and the vena cava and their branches were clearly visible in their normal relations as in an anatomical drawing. The left kidney was not visible, being covered by the descending colon and its mesentery. When lifted out of the body the intestinal mass could be twisted about at will freely in any direction. The mesentery seemed to be disposed somewhat in the form of a trough with V-shaped cross-section ending at the ileocæcal valve with the superior mesenteric artery running along the apex of the angle and in the coronal plane, making nearly a straight line for the meso-appendix in which it found its termination.

Untwisting the bowel to the left through a complete circle (360°) two bands of fibrous tissue came into view, one on each side, binding the duodenum to the transverse colon so that they were in contact. The bands were cut, care being taken not to injure the mesenteric artery and vein, and this permitted the loops of intestine to be separated an inch or more. The intestinal mass was then laid

in the abdomen in the normal relation of rotation, the ascending colon to the right, the small bowel to the left, the superior mesenteric artery passing in front of the duodenum. The tissues assumed this so-called normal position quite readily. The boy reacted normally; in fact, began almost at once to make up for his five years of starvation. He grew like the blossom stalk of an aloe, and is now a strapping, normal boy of thirteen.

MESENTERIC THROMBOSIS*

WITH A REPORT OF 6 CASES

By George G. Ross, M.D.
of Philadelphia, Pa.

Thrombosis of the mesenteric vessels is a condition of interest to the surgeon, not only because of its comparative rarity, but also because of its gravity, the difficulty of diagnosis, and the corresponding lack of success in its treatment.

Two cases of mesenteric thrombosis are noted in the records of the Lankenau Hospital in a period of ten years (1909 to 1919), during which time there were about 30,000 surgical admissions.

The anatomic points in this connection are well known and need be only hastily reviewed. The superior mesenteric artery alone supplies the small intestine and practically all of the large bowel with the exception of the descending colon, sigmoid, and rectum. The duodenum has a double blood supply. The superior mesenteric artery is stated to be an end artery, the inferior mesenteric is said not to be.

The superior mesenteric artery, then, is not only much more frequently the seat of the thrombosis, but the condition in this vessel or its branches should be correspondingly more serious than when it occurs in the inferior mesenteric area. The latter statement would be difficult of proof, because in either location the condition is of such gravity that recovery is extremely rare.

There seems to be no doubt that arterial blocking in the mesentery is far more common than obstruction of the venous circulation. Statistics have been given to show that it is twice as frequent (other authors state the ratio to be five to one).

In the reported cases there has often been no effort to differentiate between thrombosis of the mesenteric vessels and embolism. Indeed, this must often be impossible. The symptomatology is the same in either case and even at operation or autopsy it is difficult to determine whether in a given case we are dealing with a primary thrombotic or embolic condition. Venous conditions are, of course, thrombotic.

Arterial obstruction occurs either by embolic plugging of the vessel or thrombotic obliteration or by thrombosis developing at the site of lodgment of an embolus (Smith, *Wisconsin M. J.*)

Venous obstruction is said to be either of the ascending or descending variety. Whatever the nature of the beginning of the process, its course, prognosis, and treatment are the same.

There have been described also certain forms of vascular stoppage

* Read before the American Surgical Association, May 3, 1920.

more chronic in character, but all of those with which the surgeon has to deal are acute in their course.

Perhaps of more interest as a classification is the division of these cases into those in which the process is the primary one surgically; *i.e.*, the one for whose diagnosis and treatment the surgeon is called, *vide* Cases I, II, III, IV, and V, or those in which the condition follows directly after some surgical condition (Case VI), already dealt with as a complication or secondary involvement.

A great deal of attention has been given to a consideration of symptoms and diagnostic points in connection with mesenteric thrombosis. Elaborate classifications and tabulations of histories and groups of cases have failed to bring out a symptom complex upon which even a probable diagnosis can safely be made in a fair percentage of the cases seen. It is true that in some of the instances, especially those that are post-operative, slow in onset and of the venous form of thrombosis, there are no symptoms which would even lead us to suspect the true condition interfering with the patient's recovery.

A consideration of the sequence of events in thrombotic conditions will at once point out the chief fact in symptomatology and diagnosis and one practically always overlooked.

When a thrombosis occurs, the blood supply of a certain segment of intestine is stopped or diminished to a great degree. With the diminution in blood supply of such a segment there comes the natural lessening of function, manifested as lessened peristalsis. If the segment of bowel affected be other than a very minute one, peristalsis ceasing in it soon causes stoppage, due to local paralytic ileus, and we find that the case develops the signs of intestinal obstruction. Of the further changes, gangrene, perforation, etc., little need be said. They are terminal stages only.

To repeat—the symptoms of mesenteric thrombosis, in so far as they may be grouped, are the symptoms of an acute intestinal obstruction.

We have not even arrived at a point of diagnostic skill that enables us to differentiate with certainty the variety of intestinal obstruction when such an obstruction is known to exist. How much more difficult it must always be to recognize definitely the occurrence of such a rare cause of diminished or absent intestinal action as mesenteric thrombosis. But we should always be able to recognize the fact that there has taken place a grave occurrence within the abdomen demanding immediately *definitely* planned and executed *surgical* attention.

In the five cases which I have to report pain is a prominent symptom as it is in every acute intestinal obstruction.

In Case I (Germantown Hospital, January, 1919) the patient was taken sick ten days before admission with a severe attack of abdominal pain in the region of the umbilicus, and then becoming general through-

out the abdomen. Similar (?) attacks have been noted for fourteen years prior to admission.

In Case II (Germantown Hospital, February, 1919) the attack began suddenly, with abdominal pain, nausea, and vomiting.

In Case III (Lankenau Hospital, 1915) the chief complaint is given as pain over whole abdomen. It is described in detail in the history as beginning seven days before admission as an epigastric pain of gradual onset, becoming worse three days after the beginning of the pain.

In Case IV the illness is described as beginning two days before admission with pain in the right lower abdomen, soon followed by vomiting. In this case the pain remained localized in the right lower abdomen.

In Case V (University of Pennsylvania Hospital, service of Doctor Deaver) the patient was seized with a severe pain in the epigastrium.

It is evident, then, that we have in all five cases a very definite history of pain as an early symptom, in only one of the instances described as of gradual onset.

A brief consideration of the case histories themselves will make plain the fact that these are cases of obstruction not often diagnosed.

Case I.—Operated upon by my assistant, Dr. Wm. B. Swartley, at the Germantown Hospital. About ten days before admission the patient began with a severe attack of abdominal pain in the region of the umbilicus, radiation and becoming general, although worse near the midline and about the umbilicus. There was much tenderness and rigidity. The patient had not been constipated. Shortly after admission to hospital there was a peculiar looking tarry stool.

The patient states that for the last years she has had frequent attacks of severe abdominal pain, coming on suddenly, causing her to go to bed, and to be away from her duties seven to ten days. At these times the pain was located in the central part of the abdomen and in the right lower quadrant, the abdomen was sore and rigid, the bowels often constipated. She had anorexia and at times vomiting during these attacks. Such attacks occurred three to four times a year. The history otherwise is unimportant; no menstrual disturbances; one child, living and well.

Physical examination shows a fairly well-nourished woman of forty. The patient is extremely anæmic, but not jaundiced. The tongue is coated. The patient has pyorrhœa of a marked degree. Chest shows slight dullness over the apex of the right lung. No râles. The heart shows slight enlargement to left and a soft systolic endocardial murmur which is not transmitted. The abdomen is distended and tympanitic. Much general tenderness and rigidity on deep pressure and an area of dullness in the right flank suggesting fluid. No peristalsis is heard. No palpable masses or liver enlargement were found. The pain on pressure is slightly more severe to the right of the umbilicus. Vaginal examination negative. The leucocyte count on January 5, 1919, was 15,000; on January 6, 1919,

was 9000; hæmoglobin, 26; red blood count, 2,690,000. Occult blood test on fecal matter was negative on January 6, 1919, but a note on January 13th states that there were definite signs of intestinal hemorrhage. The urine showed a faint trace of albumin.

A right rectus incision was made and an appendix showing chronic obliterative appendicitis was removed. One foot of the lower portion of the ileum was found to be black and almost gangrenous, due to a thrombosis of the branch of the mesenteric artery supplying the portion of the bowel. There was a V-shaped infarcted area in the mesentery. The patient's condition did not permit of resection and the wound was closed and paitent put to bed. Under intravenous saline and stimulation the patient lived about four hours.

The appendix removed was 30 and 5 mm. The canal was obliterated. The coats white fibrous and thickened, and the appendix somewhat hooked, due to a shortening of the meso-appendix. This case shows several features of great interest. The history shows first a pyorrhœa, a possible original focus of infection. The condition of the removed appendix and the history suggest previous attacks of acute or subacute appendicitis. The heart murmur suggests a possible endocarditis and the original to be an embolus instead of a simple thrombosis of the mesenteric vessel.

The findings on admission show clearly the picture of a late stage of obstruction. The bloody stools and anæmia secondary to intestinal hemorrhage are said by some authors to suggest thrombosis. Such a marked anæmia, however, would be more likely to be taken, other things being equal, as pointing to possible malignant disease. Most striking, however, is the ten-day interval between onset and operation, rendering cure out of reasonable expectation.

Case II.—Miss C. J., aged sixty years. The patient was admitted to the Germantown Hospital February 21, 1919, having first been seen on that day by Doctor Moxey, who at once realized the gravity of her condition. The patient was sent in with a diagnosis of acute obstruction—this being entirely correct. The patient when first seen by her physician had been ill about two days. The onset had been sudden, with severe abdominal pain, nausea, and vomiting. The patient had been constipated and had without avail used both purgatives and enemata.

Physical examination showed a heavily built woman evidently very ill, in fact, in extremis. She was dyspnœic and cyanotic. The abdomen was distended and tympanitic, but not rigid. No peristalsis, the patient complained of nausea, but there was no retching or vomiting. A fatty endocarditis may have been a factor in causing the respiratory embarrassment.

Operation was undertaken as a forlorn hope. The patient died before anything could be done to relieve her condition. The small intestine, for approximately 10 feet, was found to be gangrenous, swollen, but not markedly distended. The mesenteric arteries were thickened, rigid, hard, and thrombosed, and there was no demonstrable attempt at the formation of a collateral circulation. The cæcum

and ascending colon were thickened and distended and the hepatic flexure moderately bound down by adheisons but not obstructed.

This case shows the involvement of a far greater extent of gut than Case I, a complete shutting off of the blood stream and a fulminating course. Had this patient consulted a physician at once some hope could have been entertained for her, since her condition would have been evident at any time after the onset of the disease and early operation with resection might have been possible.

Case III (Lankenau Hospital, operated on by Dr. John B. Deaver).—B. B., aged forty-six years, admitted February 13, 1917. Two days before admission the illness began with pain in the right lower abdomen. The pain continuing, after a few hours the patient began to vomit. He was given purgatives, but the patient's bowels did not move and the pain continued more or less constantly up to the time of admission. The patient complained of no fever or chill, but showed marked anorexia. His previous medical history notes: "Two previous attacks—one a month ago, and six months ago."

On admission the patient is seen to be fairly well nourished. The abdomen was somewhat distended with a tumor-like fullness in the right iliac fossa. There was rather marked tenderness and rigidity in the lower right abdomen where a mass was vaguely palpable. Peristalsis was present in the upper and left portions of the abdomen. Rectal examination revealed distinct tenderness to the right and very slight tenderness to the left side.

Operation—*four days* after admission—February 17, 1917. Under ether anæsthesia. A McBurney incision was made and enlarged upward; at a point a hand's breadth above the cæcum in the ascending colon, partial necrosis was evident, and on slight manipulation the bowel wall gave way and fecal material poured out into the field of operation. A glass drainage tube was placed in the pelvis and purulent fluid evacuated. A large rubber tube was placed in the rent in the ascending colon and the bowel closed about the tube and then sewed to the parietal peritoneum. Gauze was packed about the tube.

Peritonitis steadily became more marked after the operation and the patient succumbed.

Post-mortem inspection through the wound showed thrombosis of the mesenteric veins leading to the ascending colon.

The salient points of the history here cited are few but important. They are: (1) The simulation of an attack of acute appendicitis. The history is not typical but was sufficiently deceptive to have caused the postponement of operation for four days and the employment of a McBurney incision. (2) The early localization of symptoms correctly indicating the position of the abdominal lesion itself.

The operative procedure—drainage alone—was the correct one, the outlook hopeless at the time of operation.

Case IV.—G. A., aged fifty-two years (Lankenau Hospital, case of Dr. John B. Deaver). Admitted October 22, 1915; died October 23, 1915. The patient's illness began seven days before admission with

pain in the epigastrium, gradual in onset. Purgatives were given and the patient's bowels moved freely. Three days after the onset of the illness the patient seemed to get worse and the whole abdomen became painful. The patient began to vomit dark material and vomited everything taken by mouth thereafter. No one spot could be given as the seat of the most intense pain. There was no jaundice or chill.

The previous history mentions frequent attacks of indigestion and the use of alcohol.

Physical Examination.—The patient is a very large man, evidently in great pain. Complexion sallow; tongue heavily coated. The abdomen is greatly distended and generally tender, this tenderness being more marked in the epigastrium and left lower quadrant. Peristalsis absent. Blood-pressure, 125–80.

The patient died twenty hours after admission, not being operated on. Autopsy showed mesenteric thrombosis with gangrene of the proximal four to six feet of ileum.

A rather concise history here shows an obstruction with a typical symptom at the onset unrecognized. Three days after the onset the severe obstructive and peritonitic manifestations render it evident that operative intervention could have accomplished nothing.

Case V.—D. H. G., aged fifty-one years (University Hospital, operated on by Dr. John B. Deaver). Admitted September 25, 1918. The day before admission the patient was seized with a severe pain in the epigastrium. In the course of an hour or so this pain became generalized, affecting both the upper and lower right quadrant. The pain was paroxysmal in character, leaving the patient with a dull ache between the paroxysms. One such paroxysm of pain lasted an hour. The patient has vomited several times, once following a dose of magnesium sulphate and again following a dose of mustard water. No fecal vomiting. Bowels have not moved since beginning of illness.

Past medical history is unimportant as bearing on the present illness. Physical examination: The abdomen is tender and rigid in the epigastrium and the right iliac regions, the point of maximum tenderness being in the left upper quadrant. Little, if any, abdominal distention was present, but the upper abdomen was tympanitic on percussion. Auscultation shows peristalsis of an exaggerated and gurgling type. Blood examination: White blood count, 13,280; 76 per cent. polymorphonuclears.

After admission an enema was given with but slight result and no relief of symptoms. Lavage disclosed gastric contents having a decidedly fecal odor and appearance. A diagnosis of intestinal obstruction was made and immediate operation performed.

Operation by Dr. John B. Deaver. Ether anæsthesia. A right rectus incision was made. No mechanical obstruction was found, but there was a thrombosis of a branch of superior mesenteric artery supplying a segment of the ileum. There was considerable hemorrhage into the mesentery and a small amount of free blood in the

abdominal cavity. The segment of bowel affected was in fair condition, apparently being taken care of by the collateral circulation. Doctor Deaver expressed the opinion that no further surgical procedure would benefit the patient, and the operation was terminated. During the operation the patient received 750 c.c. of salt solution intravenously.

The first two days after operation were somewhat stormy for the patient, but after this he rallied and made an uneventful recovery.

He was discharged on the fourteenth day after operation, in very good condition. When last heard of through the family physician, he had had no return of symptoms.

This, the only case operated upon that recovered, is of note in several ways: (1) The correct diagnosis of intestinal obstruction, was made early and operation was performed at once. At operation good judgment based upon experience saved the patient from an extensive and uncalled-for procedure. The case proves, upon the living patient, a fact noted at autopsy; namely, that the collateral circulation, perhaps more often than would be supposed, has overcome the effects of mesenteric thrombosis.

CASE VI.—Mrs. C., aged thirty-seven years (Germantown Hospital). This patient had been operated upon for a pelvic condition. Intra-abdominally a cyst of the left ovary was excised, the appendix removed and the round ligaments shortened by the Gilliam method. There was also done a trachelorrhaphy and colporrhaphy, and perineorrhaphy. For six or seven days her convalescence progressed favorably. She had been catheterized regularly. Upon one occasion the nurse, after having by mistake introduced the catheter into the vagina, made the error of introducing it into the urethra without resterilizing it. The following day the patient had a well-developed septic cystitis; with a rise of temperature, chill and frequency of urination, with severe burning pain. Forty-eight hours later she had another chill with phlebitis of the left saphenous vein. The phlebitis continued an upward course into the iliac veins with involvement of the inferior mesenteric veins—through the middle hemorrhoidal vein which is the avenue of communication between the systemic and portal circulation. Her abdomen became distended and tympanitic and extremely tender. There was intense pain, nausea, and vomiting. The bowels were moved by enema and at no time did she show signs of intestinal obstruction.

Blood culture showed a colon bacillæmia. The diagnosis of thrombosis of the inferior mesenteric veins seems justified by the symptomatology and the sequence of events, although positive corroboration must be lacking because of the patient's recovery without a second operation.

A careful consideration of the foregoing case reports and of the numerous similar cases on record would lead us to a number of definite conclusions. They may be summarized as follows:

1. Arterial mesenteric thrombosis is a lesion causing a form of acute

intestinal obstruction, rare, but occurring with sufficient frequency to make it imperative to remember its possible occurrence.

2. Its symptom complex is that of an acute intestinal obstruction, slower in onset than the purely mechanical forms of acute obstructive ileus (adhesion, volvulus, etc.).

3. Venous mesenteric thrombosis is a condition of vaguer symptomatology and slower course than that formed in arterial obstruction. It tends more to spontaneous cure, and is more likely to be a secondary or post-operative condition. When, however, its remedy by the establishment of collateral circulation does not occur, it gives the same final symptoms as does the arterial form of obstruction.

4. The treatment of mesenteric thrombosis is the treatment of any form of acute intestinal obstruction—early operation. The procedure employed must vary with the condition found at operation.

(*a*) If the vitality of a segment of gut has been gravely affected, resection is indicated.

(*b*) If the patient's condition contra-indicates resection, the gut should be drawn out of the abdomen, fastened to the edges of the wound and a Paul's tube introduced, resection to be performed later.

(*c*) In the one case of this series that recovered nothing was done to the intestine and spontaneous cure resulted. While it is true that this may at times occur, and the judgment of the operator may indicate such a course, such isolated instances do not refute the general rule of early, radical procedure.

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